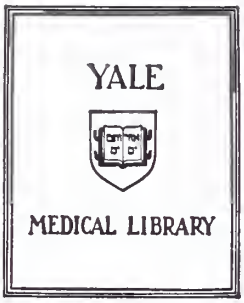
YALE MEDICAL LIBRARY
3 9002 08676 0684

WORKERS' HEALTH IN CONNECTICUT: AN HISTORICAL ANALYSIS OF THE OREIEK OUTBREAK

DAVID E. COWAN

1993


YALE
MEDICAL LIBRARY

Permission for photocopying or microfilming of "Worker's Health in Connecticut - An historical Analysis of the Uretek Outbreak"

(Title of thesis)

for the purpose of individual scholarly consultation or reference is hereby granted by the author. This permission is not to be interpreted as affecting publication of this work or otherwise placing it in the public domain, and the author reserves all rights of ownership guaranteed under common law protection of unpublished manuscripts.

Signature of Author

2-3-88

Date

YALE MEDICAL LIBRARY
Aug 26 1988

Digitized by the Internet Archive
in 2017 with funding from
Arcadia Fund

https://archive.org/details/workershealthinc00cowa

WORKERS' HEALTH IN CONNECTICUT: AN HISTORICAL ANALYSIS OF THE URETEK OUTBREAK

A Thesis Submitted to the Yale University School of Medicine in Partial Fulfillment of the Requirements for the Degree of Doctor of Medicine

by

David B Cowan

1988

med. lib
T113
H12
5592

ABSTRACT

Workers Health in Connecticut:
An Historical Analysis of the Uretek Outbreak

David B Cowan

1988

In the fall of 1986, the largest reported contemporary outbreak of occupationally induced liver injury in the United States was reported at a New Haven factory called Uretek. The plant produced polyurethane coated textiles used in rafts and inflatable aircraft slides with common chemicals employed in thousands of other plants worldwide. A clinical and epidemiological study of the plant revealed that 36 of 58 (62%) workers tested had elevations of either aspartate aminotransferase (AST or SGOT) or alanine aminotransferase (ALT or SGPT). Only 1 of 12 nonproduction workers showed any elevations ($p < .01$). Common infectious causes of hepatitis were excluded serologically in all but two workers, and toxic liver injury was confirmed histologically in five biopsied workers. The occurrence contradicted the prevailing medical opinion that toxic hepatitis was an entity of the past. The workplace survey identified Dimethylformamide (DMF), a widely used solvent and known hepatotoxin, as the most likely cause of disease. Case Reports published since the Uretek outbreak suggest that DMF may be a health concern for other workers and the public.

The outbreak was greeted with horror and astonishment by the press and government officials. However, the investigation that followed revealed that state and federal government bureaucracies had known about some hazards at Uretek for almost a decade. More important, the original furor over who was to blame for conditions at Uretek obscured a startling fact: no contemporary institution had the authority and expertise to evaluate and correct workplace health hazards like those uncovered at Uretek. Contrary to popular belief, the Occupational Safety and Health Administration (OSHA) has never been able to accomplish this task. The focus of the news reports also ignored important history: that the Connecticut Department of Health had performed just such a role as a workplace health watchdog in the 1920's until the 1950's.

The assumption that the outbreak represented an unaddressed gap in a seamless program was inaccurate and shortsighted. Uretek exposed the flaws of the current system in a uniquely lucid fashion. This paper attempts to glean from the Uretek example, and from the history of occupational health in the state, better means of protecting workers from occupational disease in the future.

Acknowledgements

I dedicate this thesis first and foremost to the Uretek workers. As is often the case in medicine, their misfortune afforded me not only an unforgettable learning experience but a topic for this thesis. I know that the Uretek experience has left its mark on many: the clinic staff, the workers, the owners, government officials, and myself. I write this in the hope that the changes wrought in the factory, and in the hearts and minds of all of those involved, will be matched by those yet to be made in our laws, agencies, legislators, and countless other workplaces.

I am greatly indebted to my friend and teacher, Dr Mark Cullen, who has nurtured my interest in occupational medicine, provided encouragement and helped me to develop a realistic appraisal of my strengths...and weaknesses. Special thanks also go to Dr Martin Cherniak who read many drafts of this effort and who provided invaluable and detailed suggestions for its improvement. I thank Dr Carrie Redlich, whom I had the pleasure to work with collecting data for the epidemiological survey, whose analysis freed me to cover other topics in this study. Finally, I would like to thank the many people who helped in other ways: Danny Perez, Beth Lewis MSW, Claudia Schmidt, Lynna Stone-Infeld, Judy Sparer, Bill Beckett MD, Lora Fleming MD, Elizabeth Coonrod, and Margery Cohen.

If the recording intern would only treat the poison from which the man is suffering with as much interest as he gives to the coffee the patient has drunk and the tobacco he has smoked, if he would ask as carefully about the length of time he was exposed to the poison as about the age at which he had measles, the task of the searcher for the truth about industrial poisons would be made very much easier.

Alice Hamilton
Industrial Poisons in the US (1925)

Between the citation appeal system, the possibility of long abatement periods, and the variance system, the clever employer can keep a modern day sweat shop humming for literally years before he feels the minor pinch of an insignificant civil fine, or at worst a suspended criminal sentence.

Leonard Dube
Connecticut UAW representative
testifying at state hearings on
February 9, 1973

...it is like something out of a Dickens novel, something out of the 19th century.

Connecticut Attorney General
Joseph I. Lieberman speaking to
the New York Times about Uretek
April 13, 1987

TABLE OF CONTENTS

CHAPTER ONE: INTRODUCTION 6

CHAPTER TWO: THE OUTBREAK 15

CHAPTER THREE: DMF AND THE WORKPLACE 45

CHAPTER FOUR: THE STRIKE AND THE GOVERNMENT RESPONSE . 52

CHAPTER FIVE: THE HISTORY 60

CHAPTER SIX: PROGNOSES AND PRESCRIPTIONS 105

BIBLIOGRAPHY 117

CHAPTER ONE: INTRODUCTION

In the fall of 1986, the largest reported contemporary outbreak of occupationally induced liver disease in the United States was discovered at a New Haven factory. The plant produced polyurethane coated textiles used in life rafts and inflatable aircraft escape slides, among other things. The coating mixture included chemicals employed in thousands of other plants worldwide. It was a gripping story of successful factory owners, irresponsible at best, and of workers, physically and economically vulnerable, and the press lost little time trumpeting the drama. From the point of view of journalists, the outbreak of liver disease, coupled with a major building collapse in Bridgeport, placed occupational health on the front page. From the point of view of the academic physicians and professionals involved, it was a textbook case of disease identification and prevention which resulted in a successful medical intervention, full recovery of most affected individuals, and a credible surveillance program to guard against recurrence. Finally, for government regulators, a potentially hazardous chemical was identified along with laws that didn't help, surveillance that didn't work, and enforcement that was timorous. No one doubted that the Occupational Safety and Health Administration (OSHA) represented a major improvement

over the unregulated past which had preceded it. There seemed to be general agreement that whatever problems existed were reflections of underfunding and understaffing. However, there was also a story that was not told and assumptions that were not questioned. Absent in the din of press accounts was an awareness of the vigorous occupational health surveillance system that existed in Connecticut in the 1940's and '50's. In the rush to assign culpability, and avoid responsibility, there was no awareness of the fact that the outbreak probably couldn't have happened 40 years ago. The hubris of the "progress mentality" caused many to miss what the workers understood: there was something terribly wrong with the system far beyond the funding of Occupational Safety and Health Administration. This thesis is an attempt to discover what lessons the outbreak, and the history of workplace surveillance, hold for occupational medicine. The next chapter presents the findings of a clinical-epidemiological survey of the Uretek workers, the first contemporary cross-sectional epidemiologic study to link hepatitis to an organic solvent.[1] Chapter three consists of a discussion of the chemicals used at Uretek, especially DMF and its known hazards. Chapter four includes a brief discussion of the strike and the response of government to the crisis, while the history of occupational health in Connecticut is the topic of the fifth chapter.

The sixth and final chapter will present recommendations for the future.

The Outbreak

Medically, the hepatitis outbreak was of interest because of the high attack rate--36 of 58 (62%) workers tested; because of the severity and chronicity of several of the cases; and simply because it occurred at all. The rarity of acute liver disease as a consequence of workplace exposure is widely accepted. The foremost authority on hepatotoxicity, US pathologist Hyman Zimmerman, concluded in 1978 that "occupational exposure has, in the past, led to subacute and acute chemical, hepatic injury. [However], **this overt form of occupational hepatic disease has largely disappeared [italics mine].**"[2] Thus, the discovery of the outbreak was not only a contradiction of the prevailing diagnostic opinion, but an opportunity to explore the detection, treatment, and natural history of a supposedly rare disease. An epidemiologic study was undertaken by the staff of the occupational medicine program of Yale medical school and Yale-New Haven Hospital.[3] The occupational nature of the outbreak was confirmed by ruling out other causes of hepatitis in the workforce and Dimethyl Formamide (DMF) was identified as the most likely cause, by reviewing biological testing data and conditions in the plant. DMF

($HCON(CH_3)_2$) is an excellent and versatile solvent widely used in the production of synthetic fibers, polyurethane, pharmaceuticals, herbicides, and numerous other compounds. Appropriate changes were made, under the direction of the occupational medicine program, to ensure that the workers safety was protected and that a medical monitoring system was established.

Social & Political Consequences

The Uretek outbreak, named for the factory where it occurred, is worthy of careful analysis for more than the unique medical information it imparts. It was a medical event with social consequences, reflected in the fact that the outbreak was the nidus for a great deal of activity apart from the epidemiological study. Uretek was a social, political, and economic drama as well. The workers were poor immigrants from Puerto Rico, El Salvador, and Chile. Some were here illegally, and possessed a paradoxical fear that recognition of the outbreak might lead to their deportation. The workers became the focus of a bitter unionization drive during the investigation of the outbreak, a drive based primarily on health and safety concerns. Meanwhile, the workers, who in the best of times were compensated near the minimum wage, experienced a type of wage intimidation all too common in the workplace: company attorneys tied the

workers' compensation cases up in court. The owners had other legal troubles having been the first company convicted of violating Connecticut's new Hazardous Waste Disposal Act. The grounds of Uretek, where neighborhood children occasionally played, were strewn with hundreds of unmarked and leaking barrels filled with waste solvents. The factory had been pumping hundreds of tons of solvent fumes into the air of a residential neighborhood for years, and with the new revelations about the factory, neighbors were angry. Everyone from doctors to neighbors, from workers to local politicians called for "the government" to do something, though this "something" was vitiated by competing interests. Shutdown was, for example, the neighbors choice, whereas the workers wanted jobs with diminished risk. This story, with the allure of life-threatening disease, competing interests, desperate immigrants, and bureaucratic inertia took the top spot on local television for days, and was front page newspaper material for weeks. Eventually it reached the national media, print and electronic.

The federal Occupational Safety and Health Administration (OSHA), department of health, department of labor, and state environmental protection agency became involved. All of these agencies expressed shock and indignation at the first reports, but in the ensuing discussions, the hepatitis outbreak somehow fell outside of each one's purview. The workers were understandably puzzled, the attorney general

was frustrated in his attempts to determine who had authority over the health of the workers, and everyone else was busy pointing fingers. "How could Uretek happen in 1987?" asked the attorney general of Connecticut.

With surprising clarity, the outbreak revealed a number of important facts about the condition of occupational health in Connecticut, and by implication, the U.S. It pointed to a profound misunderstanding of health and safety laws: misunderstanding by the public, and elected officials, of the capabilities of the agencies and misconception by the agencies of their own responsibilities. The duration of poor conditions at Uretek, at least a decade, suggested that there might be large numbers of similar "outbreaks" unrecognized and unreported nationwide. In fact, the poor conditions at Uretek persisted for so long that one wonders if they would ever have been corrected had it not been for a series of happenstances. The fact that as many as 32 of 36 workers with liver abnormalities were asymptomatic, far from confirming the rarity of occupational liver toxicity, pointed to the possibility of many undetected cases. The Attorney General's question seemed worthy of further study.

The press concluded that Reagan era budget cuts had allowed Uretek to exist and persist. OSHA had after all inspected the plant in 1979, but not since. But on deeper analysis Uretek heightens concerns, voiced for years, about

the adequacy of the Occupational Safety and Health (OSH) act and the state laws which regulate the workplace. The failure of the "system" in the Uretek case elucidates that what has become widely accepted as the answer to workplace injuries and illnesses (by management and some unions), the OSH act of 1970, is really ill-equipped to deal with disease even in the best of circumstances. The flaw, in a word, is that neither physicians nor other health professionals are involved in workplace evaluation.

A look at the uncharted history of occupational health in Connecticut reveals that workers in 1947 may have been better protected than workers in 1987. For example, physicians played a central and active role in workplace health protection through the state Bureau of Industrial Hygiene. Sadly current proposals for improving the health of workers, such as the establishment of a reliable reporting system, or a system of medical monitoring, were debated in the Connecticut legislature in 1927. Even the arguments advanced in the debate have changed little. Blaming the victim is as popular in 1987 as it was in 1927, though owners and legislators are less likely to state this view openly. In 1927, a congressman asserted that "98% of the accidents are due to the carelessness of the operator."[4] Sixty years later, Uretek attorneys maintained the position that worker illness was due to carelessness, self-abuse, and "moral turpitude". The poverty of this view is also

unchanged with time and will be evident after a review of the outbreak. Chapter five as already mentioned, examines the history of occupational health legislation in Connecticut, and in the nation to the extent necessary to make sense of events in the state.

There is at present a restricted but vigorous debate occurring about the means to improve workplace health and safety. Chapter six, Prognoses and Prescriptions, is a contribution to this dialogue. It reexamines the lessons of the outbreak with an eye to detecting and preventing workplace illness in the future.

Endnotes

1. Døssing (1986), 132.

2. Zimmerman (1978), 315. See also Mitchell, J.W. et al. (1987), 266 which appears in chapter 2.

3. Due to difficulty obtaining the cooperation of the factory owners, the study began after the institution of environmental controls made on the program's recommendation.

4. Public Health and Safety Committee Hearing (1927).

CHAPTER TWO: THE OUTBREAK

The Uretek outbreak, its size and severity, raises serious questions about the veracity of conclusions reached by Dr Zimmerman, and others, concerning the rarity of occupational liver disease. A statement issued by the American Occupational Medicine Association's Committee on occupational medical practice, "representing the composite best judgement of its members", demonstrates just how widely this view of hepatotoxins is held.

> Many of the solvents used in the early days of the chemical industry were hepatorenal toxins of some note, eg Carbon Tetrachloride. However, as this problem was recognized, solvents intended for routine use were selected so that hepatotoxicity is not a prominent feature.[1]

Although this thesis argues that the prevailing opinion is ill-founded, it is important to keep it in mind when assessing the skill and acumen of the medical professionals presented with the outbreak. Investigators had to overcome their intimate acquaintance with an authoritative view which denied the disease they were observing.

An index case is in some ways as revelatory about medical reasoning as it is about pathology. As the first patient's story is quite informative, particularly in regard to how such outbreaks can be discovered-or missed- it is presented in some detail. The rest of the cases will be

presented in aggregate and the medical and epidemiological findings summarized.

Roberto Betancourt[2], a robust 40 year old Puerto Rican, arrived in New Haven just four months before his hospital visit. He spoke little English and had left his immediate family behind. He had come to New Haven with hopes of a better life, and his brother-in-law, a worker at a local factory, had found him a job with his employer two weeks before his trip to the hospital. On the last Sunday in September of 1986 Mr. Betancourt had his niece take him to the Yale-New Haven hospital emergency room because of abdominal pain, headache and nausea that had begun a few days before. He was seen by Heidi Miller, a Physician's assistant, and Dr. Steven Sigal, a resident in medicine. Actually it was not Mr. Betancourt's first visit with these symptoms; he had been sent home from the ER two days before with the diagnosis of flu, and a day later, gastritis. Nor was he the first Hispanic man to present with a similar story, nor even the first Uretek worker referred to the occupational medicine clinic, as Mr Betancourt eventually was.[3] However, these facts were not appreciated until many weeks later. The medical history was non-specific: headache, nausea, and variable midepigastric abdominal pain which was not exacerbated or alleviated by food, though his symptoms worsened after one beer. Mr. Betancourt denied previous illnesses and alcohol or drug abuse, stating that

he drank five to ten beers per week. As in the case of most workers, there was no special sign or symptom indicating vocational etiology. In fact, it might never have come to light if not for the fact that both providers in the ER had an interest in and knowledge about work-related disease. Prompted by their unusual interest, the examiners questioned him closely about his work. The job his brother had obtained for him two weeks before was in a "glue" factory on Lenox St in New Haven, the name of which he did not know. He worked with chemicals which smelled bad but there had not been any accidents to his knowledge. He had heard rumors of other workers who had been ill but could not provide details. This line of questioning was not pursued any further at the time as the two providers had already obtained a good occupational history for a case that did not seem likely to be work-related. After just two weeks at work, it seemed far more likely that this was something he had brought with him from home: viral, or perhaps alcoholic hepatitis, the latter as a consequence of excessive drinking, despite his denial of abuse. Finally, a gastric ulcer, cholecystitis, and cholelithiasis (the latter two diseases representing disease of the gall bladder), rounded out the likely possibilities. The physical exam was unremarkable, including heme negative stool, and clinicians felt that the diagnosis of an ulcer was unlikely.

A battery of appropriate tests was ordered, including a CBC with differential cell counts, electrolytes, liver function tests, hepatitis A (IgM) antibody, hepatitis B surface antibody and antigen, and stool for ova and parasites. Concluding that Mr. Betancourt's workplace sounded worrisome even if it was not the cause of his symptoms, Ms Miller arranged for him to be seen the following Friday at the Occupational Medicine Clinic.

The occupational clinic is located in the old hospital building adjacent to the new medical center. It was founded in 1979 by Dr Mark Cullen, a graduate of both the Yale medical school and the internal medicine residency program, with the goal of filling a need for workplace health and safety services in the heavily industrialized region around the medical center, as well as to provide training to future occupational health specialists. Dr. Cullen perceived that the passage of the Occupational Health and Safety Act (OSHA) of 1970, far from resolving the issues of workplace health, had only increased the need for physicians knowledgeable about the treatment and prevention of occupational diseases, particularly since all but the largest area employers did not have their own health experts[4]. The clinic operates quite independently of the rest of the medical center as Dr. Cullen's success at funding the clinic with outside grants has brought with it a good deal of autonomy. Until the Uretek outbreak, in fact, many people within the medical

center were unaware of its existence. That is not to say that the clinic was not busy. It has always enjoyed a good reputation among area unions, health and safety groups, and the few area physicians aware of it, and as a result, there have always been more patients than staff to see them. At the time of Mr. Betancourt's visit to the emergency room in the fall of 1986 there was a three to four week wait for appointments despite the fact that the clinical staff had doubled in the previous two years to include three physicians, three fellows[5], an industrial hygienist, a part-time social worker and consulting psychiatrist, an epidemiologist, and a part-time data coordinator, in addition to social work and medical students on rotation.

My first day in clinic as a medical student happened to be the day of Mr Betancourt's appointment, October 3, 1986. Dr Cullen and Dr Redlich, a first year fellow , had already heard of the patient from Heidi Miller. There was skepticism that his symptoms could be work-related given the rarity of toxic hepatitis and the short period of exposure. Nonetheless, the story was intriguing...and quickly became more so. By the time Mr Betancourt was seen in clinic he was feeling somewhat better. Ms Miller had instructed him to stay away from work which had turned out to be no problem: his employer had fired him when he called in sick. A number of laboratory results had come back: the day of his third ER visit (9/28) the Aspartate Aminotransferase (AST or

SGOT) had been 949 U/L (nl 15-35) while other tests including Alkaline Phosphatase, Bilirubin, and Lipase were all normal. Hepatitis A IgM antibody was negative as was Hepatitis B Surface Antigen. Hepatitis A IgG was positive indicating a distant history of Hepatitis A infection but this was unsurprising in a Puerto Rican native and did not account for the current signs or symptoms. Repeat Liver Function Tests (LFT's), drawn the day prior to the occupational clinic visit, showed the AST greatly improved at 31, but the Alanine Aminotransferase (ALT or SGPT), assayed for the first time, at 317 U/L (nl 6-37). The patient was again asked about alcohol and drug consumption and he adamantly maintained his earlier report of no more than 10 beers per week. It was decided that an ultrasonographic examination of the gall bladder was indicated; a few days later it was reported as normal. The patients symptoms and transaminases were improving slowly and he promised to bring in the name of the factory on a follow-up visit (he had no phone) in a few days. After screens for Cytomegalovirus and Epstein-Barr virus were negative, the presumptive diagnosis of toxic hepatitis was reached, by exclusion, with the thought that perhaps the patient was peculiarly sensitive to the chemicals in the plant. Everyone wanted to find out more about the employer, but there was no outward harbinger that this was an index case.

In another stroke of good fortune, Heidi Miller again happened to be in the ER a few days later when another Hispanic gentleman arrived with the same symptoms, and importantly the same place of employment on Lenox St. The company was called Uretek, and there he and 40 others coated fabric with "glue" for parachutes and life rafts he said. The second patient was also evaluated in the Occupational medicine clinic with the same results: elevated LFT's and abdominal pain, no alcohol abuse, no infectious diseases, and no job, as he too had been fired when he reported he was ill. The clinic social worker was called in to help both workers apply for worker's compensation. Although it appeared that they had been fired illegally, legal activity was postponed; no one was eager to go back to Uretek.

Dr. William Beckett, a faculty member in the clinic, took on the chore of contacting the employer. This can be a frustrating endeavor as the Yale clinic operates without any legal authority and can only enter workplaces with permission. Although a physician or anyone else is free to contact the Federal Occupational Safety and Health Administration (OSHA), the clinic had been frustrated by the results of recent attempts. Since Reagan had taken office, OSHA had taken to inspecting only on the complaint of a worker. Betancourt and his colleague had been conveniently terminated. While logic would dictate that an inspection might be prudent, recent experience raised doubts about the

prospect of a vigorous inspection. OSHA had been known in recent years to call an employer in lieu of inspecting if the complaint had not come from a worker. A few months before, OSHA had declined to inspect a dry cleaning business from which clinic staff had examined several overexposed workers (and which refused to cooperate with the clinic) on grounds the complaint was "too vague". The clinic industrial hygienist customarily received long questionnaires in response to reports to OSHA. As she said, "if I knew the answers to all of their questions, we wouldn't have filed an OSHA complaint to begin with."[6]

Even if an inspection had been performed there were additional reasons to doubt its quality and timeliness. OSHA, badly underfunded during the Reagan years, had a long backlog of inspection requests. Morale among the remaining inspectors (only two remained in Connecticut) was low. It was understood that no one would be promoted if they were viewed as a zealot. Much more important, however, than the lack of funding was lack of expertise. Even pre-Reagan OSHA lacked the personnel to deal with a health problem such as toxic hepatitis, with no physicians involved at the inspection level. There was also always the chance that the offending substances might not be on OSHA's list of regulated hazards; only 500 have been recognized to date of the more than 50,000 substances in widespread use. On top of it all, procedural snarls could delay abatement of

enforceable hazards for months or even years. With the prospects of a bungled investigation, or worse a stamp of approval on existing conditions, and a health problem that called for swift action, it seemed wiser to delay contacting OSHA if by doing so the clinic could identify the process responsible for hepatotoxicity. To do otherwise was to risk sentencing the remaining Uretek workers to continued morbidity or mortality. There was eventually a debate among the staff about notifying OSHA and the press when the needs of the fired workers conflicted with those that still worked at the plant. For the moment, however, the public's right to know was viewed as secondary to the clinic's responsibility to the workers.

It was unclear if Dr Beckett would be successful. First he was told that he would have to speak to Mr Andrews, then a week later that Mr Andrews would be "away in Europe for a few months". Dr Beckett explained that we were concerned about a potential hazard at the plant that couldn't wait a few months: two workers had toxic hepatitis, an illness that could be fatal if exposure continued. We would need to know what chemicals were in use and would like to see the production process. He reminded them that the Yale program did not issue fines or charge a fee but would simply make recommendations. Fortunately, this approach works with most employers. Uretek however was unmoved; they would have to check with their attorney as the formulae were

"proprietary". In the face of two seriously ill patients and the companies uncooperative stance, even the unperturbable Dr Beckett was getting frustrated. When Uretek called back to say "thanks, but no thanks" Dr Beckett mentioned that he felt compelled to contact OSHA, knowing that this was probably a hollow threat, given the typical OSHA response, but cognizant that it was our only leverage. Ten minutes later, somewhat to everyone's surprise, the plant called back. A visit would be fine, they had "misunderstood" and hoped that there was no need to notify OSHA. At the time it was concluded that the employer was simply unaware of OSHA's impotence, and ignorantly timorous. Unbeknownst to anyone in the clinic was the fact that Uretek was awaiting sentencing as the first convicted violator of the state's toxic waste disposal act, and Mr Andrews was facing a possible 15 year jail term. A meeting was arranged for November 6th.

By the time that the plant visit occurred Dr Cullen had received a worrisome phone call from a clinic in the heart of the Hispanic community. They'd seen an unusual number of gastro-intestinal problems that fall, did Cullen have any thoughts? Concern about the remaining employees was running high on the cold November morning of the plant tour, and although the clinic staff had seen some gritty workplaces, Uretek was surprisingly bad. Gary Dorsey, feature writer

for The Hartford Courant, paints an accurate portrait, at least from outside:

> The factory was almost hidden by trees, sitting like an overbaked brick wedged into the eastern banks of the Quinnipiac River. From across the water it looked like a rust-colored vestige of a New England industrial era whose time had passed. Tool and Die. Cut and Sew. Weave and Spin. Stockpiles of rotting, trash-filled drums, boarded-up windows, and tangles of weeds and vines signaled abandonment.[7]

But the exterior appearance masked the intensity of activity on the inside. Much reassurance and persuasion was required to establish what chemicals were used in the plant. At first claiming that he did not know, Andrews eventually gave a list of chemical names: Toluene, Dimethyl Formamide, Chlorobenzene, and Methyl Ethyl Ketone, all used to make polyurethane or clean machinery. There was also reluctance about a walkthrough but Beckett and Sparer prevailed upon Andrews and company attorney Clifford Grandjean.

Uretek was ready for the inspection, or at least they thought they were. Employees were wearing new looking vapor masks and safety glasses, or could be seen putting them on as we walked through the plant. Trash pails were filled with the boxes the safety equipment came in and it was fairly obvious that they'd never been worn before: everything in the plant, including the workers, was covered with layers of dried polyurethane except for the safety equipment. The air was thick with organic vapor sufficient to cause a headache and light-headedness amongst those on

the tour.[8] Workers were standing by open drums of polyurethane, ladling it onto fabric travelling by on rollers. No one wore gloves. Ventilation consisted of a few window fans which were turned off, even in the shed where the urethane was mixed. The area of strongest fumes was around the ovens through which the fabric passed to dry (see photos). There were exhaust hoods over the ovens but it was clear from the smell that much organic vapor was ending up inside the shop.[9] Unfortunately Ms. Sparer was not permitted to make measurements, other than with her nose and eyes. Sparer, an OSHA hygienist before coming to Yale, stated: "It was what we'd call, at OSHA, a real pit."[10]

The walk through also revealed several other facts: all but a few workers, who numbered sixty-six, were Hispanic, and most did not speak English. Few seemed aware of the hazards at the workplace - they warmed their lunches in the solvent ovens and ate them as they ladled polyurethane onto the fabric. It was also clear that the plant had not been inspected recently. It was dark, hot, cluttered with open drums of chemicals everywhere and lacked safety showers, eyewash stations, and other rudimentary safety equipment.

A report was prepared and sent to Uretek on November 19. It recommended immediate installation of ventilation and safety equipment and a blood screening program to evaluate all employees for liver abnormalities. The

company's initial reluctance was replaced by quiet cooperation and two days later they invited physicians back to talk with workers about the blood tests. The first screening occurred in early December and on December 5th, a Friday, results trickled in from the lab: 30 of 45 individuals had some abnormality in their liver enzymes, about 33% of them more than twice normal. An isolated problem had become an unprecedented outbreak, contradicting the wisdom of Zimmerman and others. The clinic staff was more than concerned, they were alarmed. Dr Beckett phoned Uretek that afternoon at 4:45 p.m. and described the situation to the production manager, recommending that the plant shut down until ventilation equipment already ordered could be installed. The manager stated that Uretek would comply.[11]

In the ensuing months the clinic completed an epidemiological study of the workers and an environmental survey of the workplace. The results of the clinic study are presented below, while the findings of the environmental survey, primarily concerning DMF, constitute the next chapter.

Methods: Based on the cases seen in clinic, the blood drawn for the screening was tested for the following: AST, ALT, gamma-glutamyl transpeptidase, direct and total bilirubins, alkaline phosphatase, lactic dehydrogenase, total protein, and albumin. Individual liver function test results and an

interpretation, along with a questionnaire to determine demographic background, job history, and symptoms were sent to all workers in English and Spanish. Workers with AST or ALT greater than twice normal were referred to the occupational medicine clinic, along with anyone else who wished to be seen. Clinic evaluation consisted of history and physical exam, Liver Function Tests, Hepatitis A & B serologies, a complete blood count, prothrombin time and partial thromboplastin time. Additional blood tests included BUN, creatinine, glucose and electrolytes. Five had abdominal ultrasounds and liver biopsies.

Screening results: Ultimately, medical data was obtained on 58 of 66 employees (see Figure #1). As the company provided only names, but not job classification or length of employment, this information was collected by questionnaire or during a clinic visit (only 46 of 58). Average length of employment was 40 months, although 15 of 46 (33%) had worked for three months or less. The age range was from 18 to 60 with a mean of 33 years. Workers who had worked in production anytime in the preceding six months were considered production workers, all of whom were male, all but three Hispanic. These individuals worked five 12-hour shifts plus one or two 8-hour shifts per week.[12] The remaining twelve workers were classified as non-production - inspecting, packing, shipping, or office workers. A third

were Hispanic, two female. The eight individuals who did not participate were non-production personnel.

Overall, 36 or 62% of workers tested had an abnormal AST or ALT, 19 or 33% had one or more tests greater than twice normal and 9 or 16% had one test greater than fivefold normal (see Figure #2). As shown in Figure #2, enzyme elevation correlated with job classification with 11 of 12 non-production workers entirely normal, while 35 or 76% of production workers showed an elevation. Mean ALT for non-production workers was 30 ± 7 U/L versus 113 ± 171 U/L, a significant difference ($p < .01$) by Student's T-test. Of interest, the data demonstrate an inverse correlation between transaminase level and duration of employment with short-term workers showing the greatest abnormality (Figure #3). As a longitudinal study was not possible, no further information could be collected on this issue. Symptoms reported on the questionnaire were 1)GI (anorexia, abdominal pain, or nausea) in 67%, 2)CNS (headaches,dizziness) in 39%, 3)Alcohol intolerance ("disulfiram-like reaction" characterized by facial flushing, palpitations, nausea) in 24%. All workers with elevations were either removed or transferred to non-production areas. Before engineering modifications were completed, the majority of the production workers went out on strike (two months after initial screening - see next chapter).

Medical evaluation and follow-up: Four workers were evaluated in clinic before the screening and 23 following it, including all nineteen of those with enzymes twice normal. Viral serologies showed all 27 with negative hepatitis B surface antigen and negative hepatitis A IgM antibody. Two had positive hepatitis B surface and core antibody indicating immunity subsequent to a past infection. None had received blood transfusions or were using prescription medication. Only two acknowledged significant alcohol or illicit drug use. The five hepatic ultrasounds performed were negative.

The interpretation of the liver biopsies proved to be controversial. They were performed from two to eleven months after initial screening in the five cases that demonstrated persistent enzyme elevation. They all revealed microscopic changes consistent with resolving toxic injury to the liver. These included focal steatosis, spotty unicellular necrosis and evidence of diffuse regeneration, including irregular liver cell plates, binucleated hepatocytes and variation in nuclear size and staining characteristics. A fat stain of a frozen section revealed diffuse steatosis in a microvesicular pattern not evident on the conventional paraffin sections. Only one biopsy showed evidence of a chronic process evidenced by scattered acidophilic bodies, aggregates of mononuclear inflammatory cells within sinusoids, and replacement of individual

hepatocytes by inflammatory infiltrate. There was no fibrosis. However, as Dϕssing (1986) points out:

> Except for a few rare causes of hepatotoxicity such as vinyl chloride and methylene dianiline there is no specific or even typical clinical, chemical, or pathological feature of occupational hepatotoxicity. (131)

The biopsies were reviewed by a number of pathologists, including Hyman Zimmerman, the hepatic authority who had pronounced industrial toxic hepatitis a vestige of the past. He found all the biopsies consistent with a diagnosis of Non-A, Non-B hepatitis, a relatively uncommon form of viral or infectious hepatitis for which there is no test in clinical use. Non-A Non-B hepatitis is usually transmitted in North America by blood products, needle sharing, tattooing, etc. There are only a handful of epidemic outbreaks none of which occurred in the First world. Given the cluster of cases in one workplace, the lack of risk factors (i.e. IV drugs or transfusions) and the acknowledged low incidence of Non-A Non-B hepatitis Dr Zimmerman's interpretation seems unlikely.[13] Conclusions of the Yale pathologists varied widely: several felt that all biopsies showed signs of toxic workplace injury, others felt one or more represented Non-A Non-B based on particular features. It should be pointed out that, as with toxic hepatitis, the diagnosis of Non-A Non-B is not based on any published study of the microscopic appearance of various hepatitides, but only on the pathologists' own judgement. While the value of

tissue and the opinion of a skilled pathologist in reaching a diagnosis is unquestionable, it is often ascribed an importance that exceeds the limits of scientific certainty. The case of the worker's biopsy with chronic inflammatory infiltrate is illustrative. This worker, an 18-year-old with a two-month exposure history and AST/ALT of 313/582 at screening and 49/156 in October 1987 (11 months later), has been denied workers' compensation based on one pathologist's diagnosis of Non A Non B. While pathologists may recognize that the pattern of injury does not unambiguously reveal the cause of injury, and while debate is healthy, the consequences of denied compensation are far from academic for this worker. Conflicting standards of proof in clinico-epidemiological versus laboratory medicine and in the legal/administrative sphere appear frequently. Epidemiology seeks to discern causes in the pattern of clusters of cases not individuals. Pathology focuses on individual cases and is fairly indifferent to probabilistic thinking and new disease patterns until widely accepted. The courts establish cause by the definition "it is more likely than not that A caused B" which in practice can translate to A caused B, yes or no. When these three views of causation collide the outcome for the individual can be unsatisfactory and unfair.[14] The case of the 18 year old Uretek worker is being appealed.

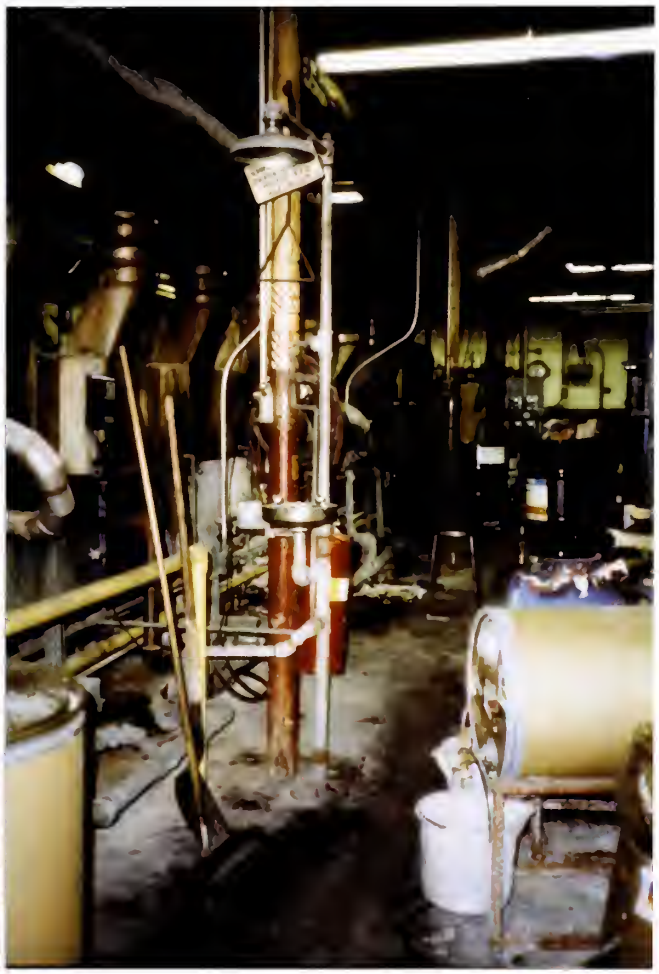
Most workers' enzymes resolved to normal levels within one to five months of removal or transfer. Eight individuals had enzyme abnormalities which persisted six months or longer and three, including the 18-year-old, remain elevated in March 1988, but all seem to be resolving, if more slowly, than most affected workers.

Discussion: The epidemiological study, while not perfect, provides strong evidence that Uretek represents an outbreak of work-related liver disease. Given the nature of the work force, largely non-English speaking and in some cases transient, and the limitations imposed by the employer, the amount of information gathered is impressive. Three factors point to a chemical at work as the causative agent: 1)the proportion affected (76% of production workers, 33% seriously) 2)the significant difference between production and non-production workers (the one affected non-production worker showed very mild elevations) 3)the lack of risk factors infectious or other forms of hepatic injury. With regard to this last point, doubts might be cast on the reliability of self-reported alcohol consumption. A study published in 1979 in Digestive Diseases demonstrated that an AST/ALT ratio of 2 or greater is "highly suggestive of alcoholic hepatitis and cirrhosis."[15] The test was particularly helpful in cases where ALT was less than 300, the moderate sort of elevation seen in most Uretek workers.

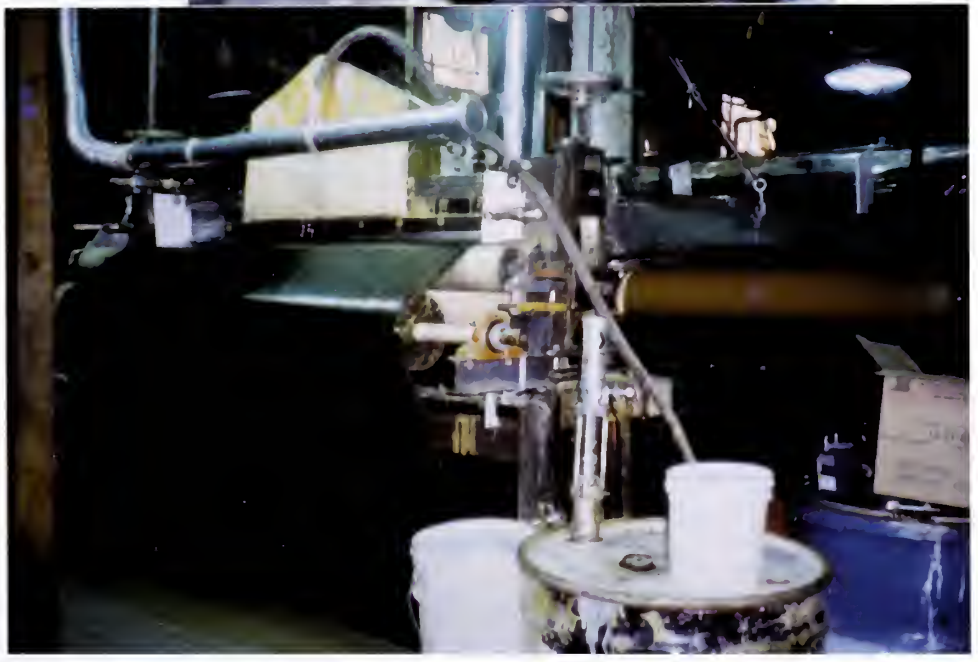
In the 1979 study, 91% of patients with alcoholic liver disease had an AST/ALT ratio greater than 1.0.[16] Figure #4 shows a graph of AST/ALT ratios for all Uretek workers with elevation - only 1 of 36 was greater than 1.0. While no biochemical assay is specific enough to allow definitive conclusions, this result does not contradict the workers' self-reports of minimal to moderate alcohol consumption (except in two cases of self-reported abuse).

Another issue which raises some doubts is the inconsistency between presumed degree of exposure and liver injury. In the absence of dosage measurements one might assume that length of employment might be correlated with illness. In the Uretek case, however, elevated liver function tests were clustered in workers of short tenure, as shown in Figure #3. There are several possible explanations. First, there is almost certainly a variability in the hepatic sensitivity to environmental toxins, though we would expect to find it randomly distributed amongst the workers if this were the sole factor responsible for the cluster of greater sensitivity in new workers. Second, there is a "healthy worker effect", that is, workers who had severe reactions left the plant, or as in Betancourt's case (and others), fired. Statisiticians call this "selection bias". The intolerance of most new workers was so well-known, in fact, that the old-timers ran a betting pool to see who could predict how long a new man would last. This could explain the presence

of highly sensitive individuals in the recently employed group, while veterans showed much milder elevations. The fact that no records were kept on the number of workers who quit or were terminated makes it impossible to find the missing cohort of workers who left due to illness.[17] A final possibility is that Figure #3 represents the natural history of the disease process, which involves acute injury followed by a period of gradual resolution. Which of these factors is responsible for the observed pattern and to what degree is impossible to ascertain with the cross-sectional data collected. All of the symptoms reported and signs observed are consistent with toxic injury due to chemicals used at Uretek.



WORKPLACE PHOTOS: As none were permitted on the initial visit, all of the photographs were taken after the installation of recommended ventilation equipment. The left hand photo shows the primary means of ventilation before the changes. Many of these fans did not work. The one on the right shows the apppearance of the shop after it was cleaned up and safety equipment installed.



These two photos show the polyurethane mixture being pumped from a barrel onto the fabric as it travels through rollers and on to the drying oven. The horizontal bar with flexible tubing is part of the new local ventilation system.

The top photo displays a worker wearing measurement instruments standing near oven. The bottom photo shows another worker with vapor mask standing at "new" machine (note skin exposure). Formerly this worker stood next to a barrel of polyurethane and ladled it onto the fabric "knife".

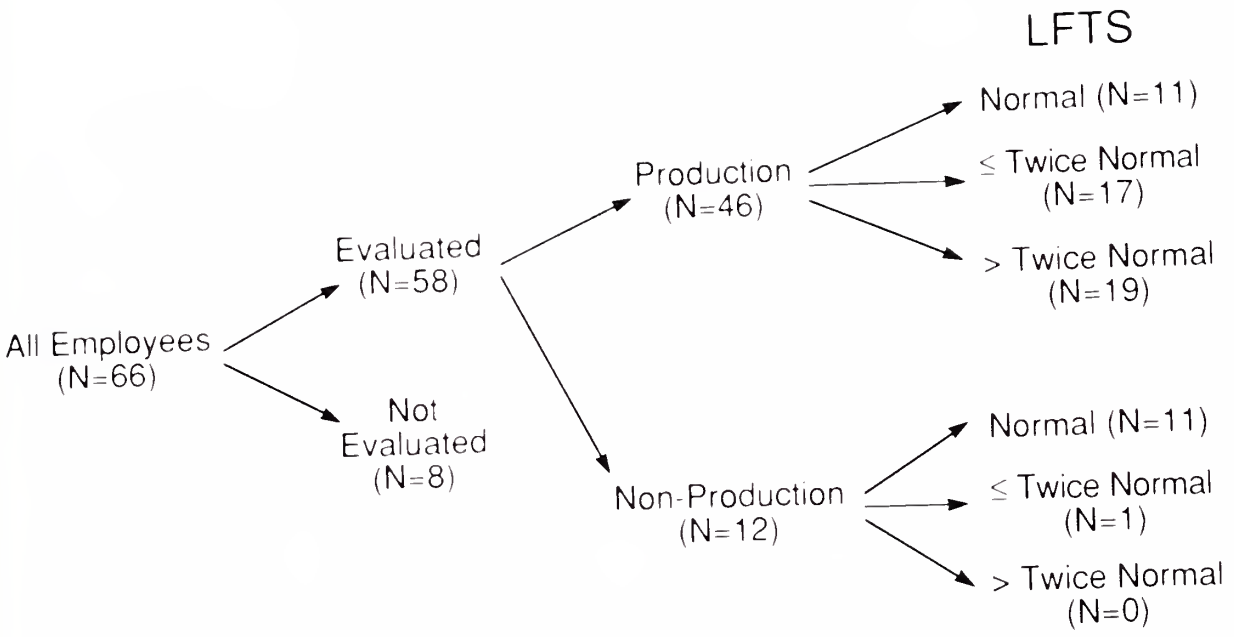


FIGURE #1: Flow chart showing LFT data obtained on the workers. LFT's were collected on 58 out of 66 total employees. 46 of these were production workers; 12 were non-production workers. Of the 46 production workers 11 had normal liver enzymes; 35 had one or more elevated transaminases. Of the 12 non-production workers 11 had normal liver enzymes and only one had mild elevations.

LIVER ENZYMES IN PRODUCTION AND NON PRODUCTION WORKERS

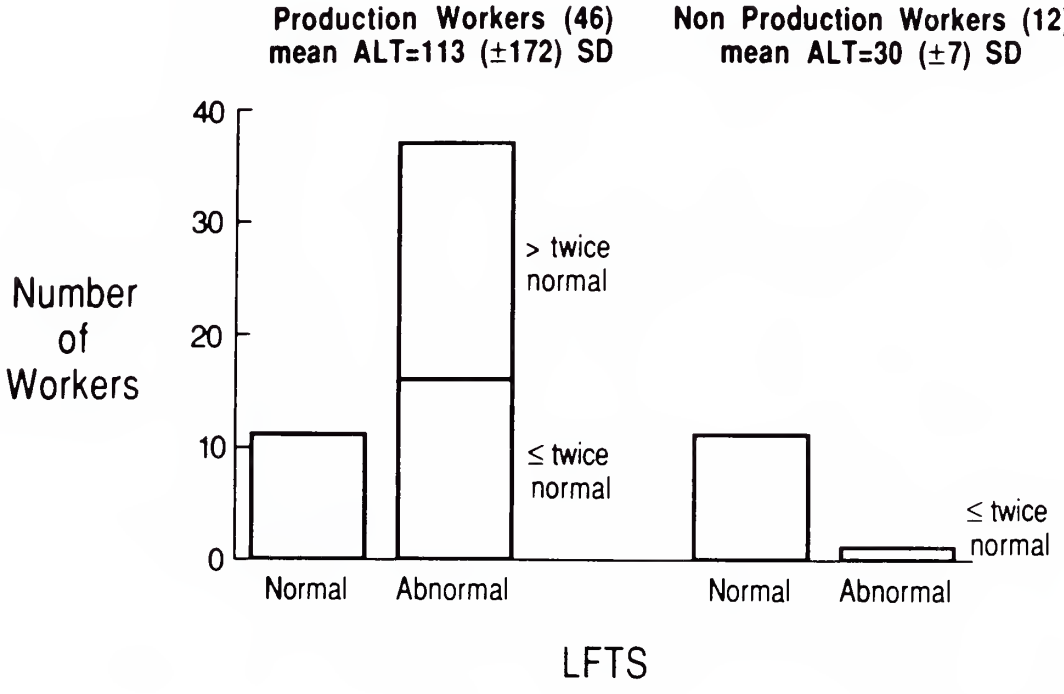


FIGURE #2: Frequency distribution of workers with liver abnormalities. The graph shows numbers of production and non-production workers with AST or ALT values greater than or less than twice normal.

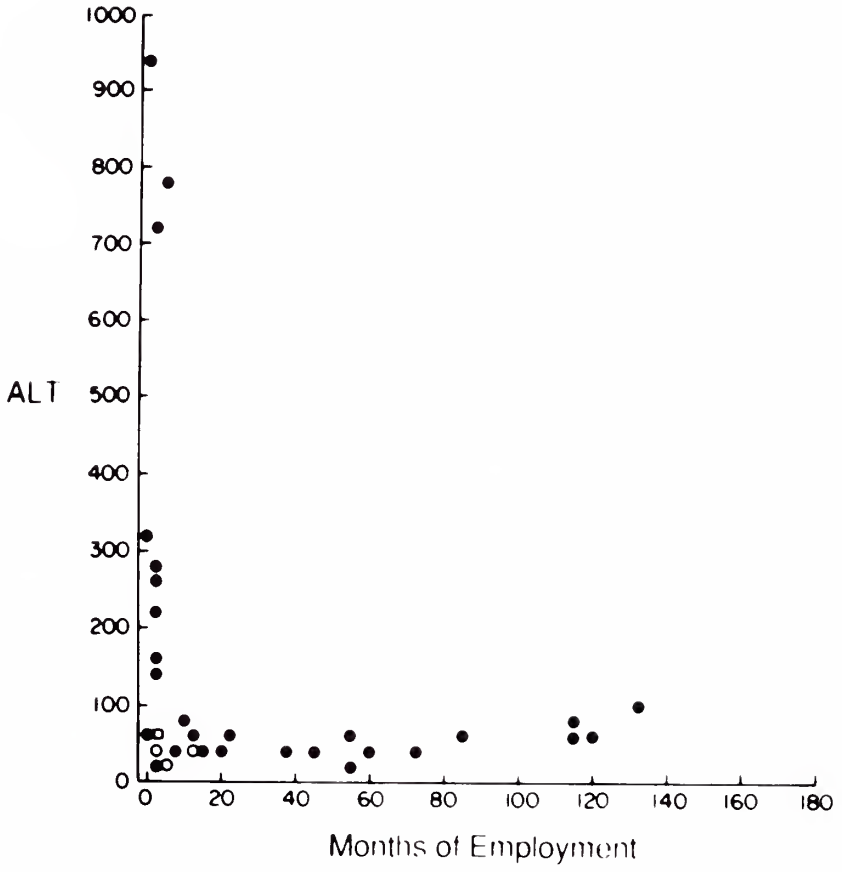


FIGURE #3: Relationship between months of employment and ALT in production workers who had completed the questionnaire (N=32). Months of employment was determined on the basis of questionnaire data and calculated from month of initial hire to the date liver function tests were drawn. ALT used was from the initial screening or from the first clinic visit if the patient had continued exposure after the screening test.

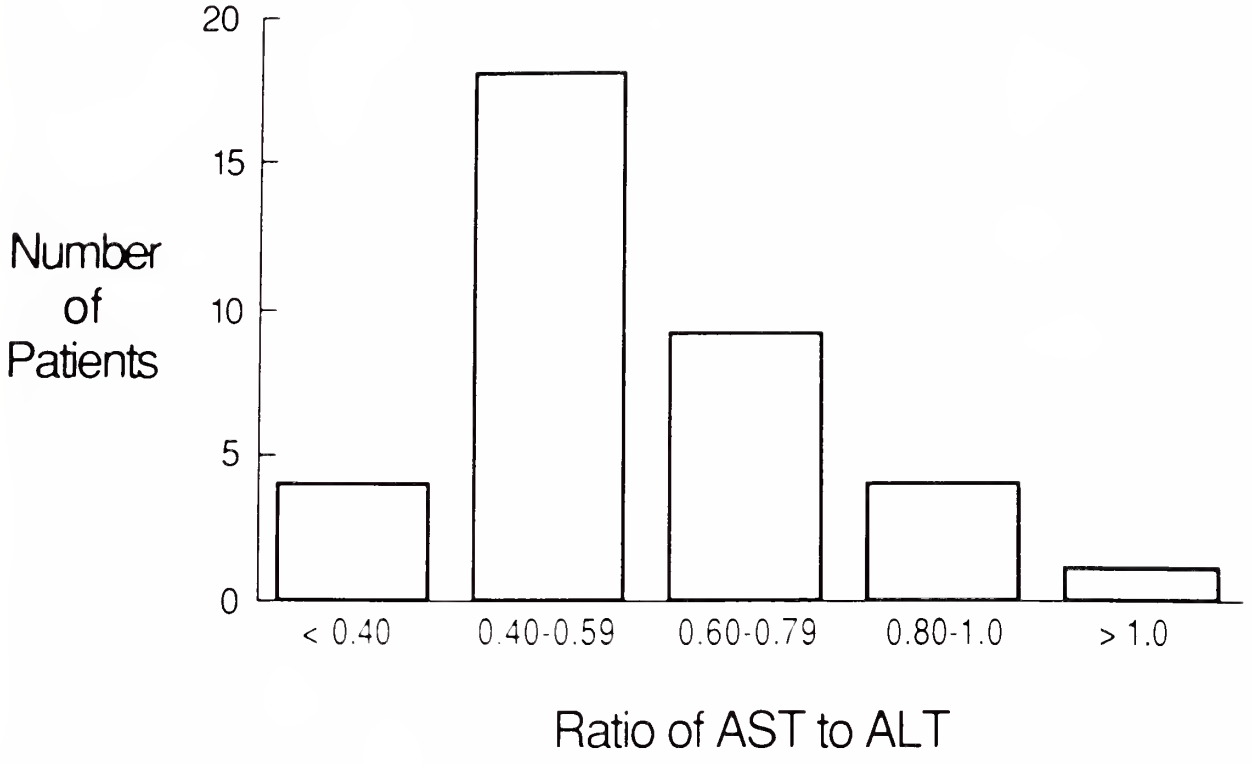


FIGURE #4: Frequency distribution of the ratio of AST to ALT in production workers (N=36) with one or more elevated transaminases. If the patient had more than one set of liver enzymes, the set with the highest AST and ALT was used. The ratio of AST to ALT was less than or equal to one in all but one patient, the opposite of the ratio normally seen in alcoholic disease.

Endnotes

1. Mitchell et al. (1987), 266. see also Dφssing (1986).

2. This is the patients actual name as his identity has been previously revealed, with informed consent, in numerous press accounts.

3. A computerized search of the clinic records revealed that an Hispanic Uretek worker was seen in 1984. No liver function tests were done and the patients symptoms resolved and were presumed to be viral gastro-enteritis. In retrospect, they probably represented solvent effects.

4. Chapter five on the history of federal and state legislation will deal more extensively with these issues.

5. Fellows have completed a three year residency training program and are seeking two to three years of sub-specialty training in occupational medicine and public health.

6. Dorsey (1987).

7. Ibid.

8. Because no air level measurements were permitted by the company, it is not known if levels exceeded the OSHA limit of 10 ppm.

9. The amount ending up <u>outside</u> qualified Uretek as a "big guy" among Connecticut polluters according to Steve Peplau, a senior air pollution control engineer with the state. Peplau estimated discharge into the air "around 300 tons per year." Quoted in Dorsey (1987).

10. Ibid.

11. Later it was revealed that Uretek kept the night shift running on unventilated machines until the strike shut the plant down in February.

12. As they were generally paid minimum wage, overtime was the major attraction of work at Uretek.

13. Dienstag et al. in Harrison's Principles of Internal Medicine, 11th edition, 1987, 1325.

14. See also Brennan & Carter (1985) Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease In Individuals and chapter six.

15. Cohen & Kaplan (1979)

16. Ibid.

17. Rumors abounded of workers who had returned to Puerto Rico where they could afford medical care. They almost certainly were not identified as victims of occupational disease.

CHAPTER THREE: DMF AND THE WORKPLACE

As previously described, Uretek was engaged in the production of polyurethane coatings and their application to fabric. Of the list of chemicals provided by Uretek, only one chemical had been identified already as a hepatotoxin, Dimethyl Formamide (DMF). While the other compounds, Chlorobenzene, Toluene, and Methyl Ethyl Ketone might have a role in the outbreak, particularly as synergists, there are only a few studies documenting hepatotoxicity, and then primarily in animals. A toxicological study will be necessary to establish with greater certainty the health effects of DMF. Like most industrial compounds, DMF has not been studied in combination with other chemicals, with the exception of alcohol. However, enough is known about DMF to make a convincing argument that it is at least partly if not entirely the cause of the hepatotoxicity at Uretek.

An excellent and versatile solvent, DMF is used ubiquitously in polyurethane products and acrylic fibers (65-75%) as well as pharmaceuticals (15-20%) and pesticides, among other applications. It is an extremely important solvent known for its excellent solubility of a wide variety of organic and inorganic compounds. Approximately one third of the world's capacity to produce acrylic fibers depends on DMF.[1] First synthesized in 1893, DMF only came into

widespread commercial use in the early 1940's. It was generally considered non-toxic by research groups associated with manufacturers until very recently.

Although US production was estimated at 33 million pounds in 1979, and worldwide output at 106 million pounds, the number of workers exposed is unknown.[2] A recent report from the occupational medicine program at Mt. Sinai hospital in New York contains an estimate of over 100,000 U.S. workers exposed to DMF currently, or in the last decade.[3] Santodonato, et al (1985), note that with the large quantity produced and the number of applications, "exposure would appear to be quite widespread."[4]

A review of the literature failed to turn up specific data on the pharmacokinetics of DMF absorption, except for a Japanese study indicating that trans-dermal absorption is significant.[5] There is consensus that respiratory and gastric absorption occurs. DMF is metabolized to N-Methylformamide (MMF) and N-Hydroxymethyl-Methylformamide.[6] In humans exposed to DMF, N-Methylformamide has been reported as the principal urinary metabolite and has been proposed, along with elevated urinary mercaptopurines, as a means of biological monitoring in workers.[7] Studies demonstrate a directly proportional relationship between total exposure and urinary MMF, though MMF fell to undetectable levels within 14 hours of exposure.[8] Spot urines do not correlate well with exposure, however, and there is

great variability if urine levels in exposed individuals. Some critics of the Uretek study have argued that urine testing should have been done to establish DMF as the causative agent. Measurement of MMF excretion in Uretek workers would have required 24 hour urine collection and would have helped to establish DMF as the causitive agent. However, the conclusions would not have been altered as workers had tangible dermal and respiratory exposure sufficient to account for the findings.

Toxicological studies have established that DMF is hepatotoxic in cats, mice, rats, guinea pigs, and rabbits, causing a centrilobular necrosis and fatty degeneration.[9] Reports involving humans have been surprisingly few and generally involve one or two cases in a given workplace. All but one case reported occurred outside the United States.[10] All the human studies document abdominal pain, nausea, vomiting, dizziness, headaches, anorexia, diarrhea, and, in a few cases, agitation. Liver function tests were rarely evaluated except in two studies which documented elevations after large accidental exposures.[11] The "disulfiram-like" effect of DMF has long been recognized and is attributed to possible acetaldehyde inhibition by MMF.[12]

The ubiquity and high toxicity of DMF poses the question of why so few cases of hepatic injury appear in the literature. The lone U.S. case report occurred in a Pennsylvania Urethane coating plant.[13] Conditions at Uretek

certainly may have been worse than average, and the recommended precautions may be better observed in other operations (manufacturers now recommend skin and respiratory protection). Even the Pennsylvania plant had instituted periodic LFT assays as far back as 1972. However, Santodonato, et al (1985), points out that solvent applications of DMF, such as cleaning of machinery, "are likely to be conducted with few or no engineering controls, thus undoubtedly resulting in worker exposure." The National Cancer Institute report concludes prophetically with the following paragraph:

> There are no reports of well-designed epidemiologic studies on worker groups exposed to Dimethylformamide. Case reports from the foreign literature have associated DMF exposure with increased morbidity from various causes. Worker exposure to DMF during acrylic fiber production is apparently sufficient to justify consideration of this cohort for further investigation. Exposures of similar or greater magnitude may occur in other industries (e.g. pharmaceutical production, chemical synthesis), although cohorts are likely to be small and simultaneous exposure to multiple chemicals is likely to occur.

Tolot (1968) concluded that:

> The frequency of these symptoms is probably quite widespread, probably greater than one might anticipate in view of the relative silence which surrounds the question in France, a silence which should be deplored.

With Uretek, the silence in the U.S. surrounding DMF has been broken. There are signs that DMF and methylamines are already subjects of national interest and controversy.

The Uretek outbreak coincided with the publication of the first adequate studies of DMF carcinogenicity. Prior to 1986, short-term mutagenicity and genotoxicity studies were negative and an animal bioassay was in progress, One study reported chromosome damage in DMF workers, but the health significance had not been established.[14] The news since Uretek has not been good. An unpublished 32-year prospective study of 5,000 workers by a DuPont epidemiologist released in September 1986 documented significant excesses of bucco-pharyngeal and malignant melanoma in DMF-exposed cohorts, and an excess of prostate cancer in a cohort exposed to DMF plus Acrylonitrile (ACN).[15] In October 1986 a report appeared in the Journal of Urology describing clusters of embryonal cell carcinomas of the testis in two separate aircraft repair facilities among mechanics who had worked extensively with DMF.[16] Fourteen months later, in December 1987, a letter appeared in The Lancet describing three cases of testicular cancer in tannery workers exposed to DMF. Since then two additional cases have been identified by researchers at Mt Sinai Medical Center in New York. The Mt. Sinai researchers and a labor union petitioned federal agencies to issue a bulletin warning workers of potential carcinogenicity of DMF.[17] All of these recent developments point to even greater hazards from DMF than the risk of hepatotoxicity highlighted by the Uretek outbreak.

Uretek workers are being informed of these recently reported risks by the Yale clinic staff.

A coincidental report received by the author indicates that there may even be a DMF hazard to the general public. Ellen Silbergeld, PhD senior scientist for the Washington-based Environmental Defense Fund, reports that the EPA has recently licensed an algicide/herbicide which breaks down into MMF. This product, marketed as Sonar (Fluridone), is not the only licensed cidal agent which includes MMF as a degradation product. However, the EPA felt that Fluridone is sufficiently safe to justify use as an algicide in drinking water reservoirs. The township of Lake George, New York, has already attempted to employ it for this purpose, though it is being opposed by citizens' groups who called on Dr Silbergeld to testify. The EPA has declined to review its classification in light of the new cancer reports even though they acknowledge that they were not aware of them when they approved the product. Dr. Silbergeld has recommended that the EPA's research arm, the NIEHS, undertake a new study of Fluridone. Clearly the safety of DMF and its metabolites will be significant issues in the coming months and years.

Endnotes

1. Santodonato (1985)

2. Eberling (1980) quoted in Santodonato (1985). According to the Bureau of National Affairs (1987), there are four U.S. manufacturers: Air Products, DuPont, Redell, and Hall.

3. Ducatman et al (1986). The estimate is based on an as yet unpublished NIOSH survey.

4. Ibid.

5. Ibid.

6. Lundberg et al (1983).

7. Barnes & Ranta (1972).

8. Krivanek et al (1978), Barnes & Henry (1974), and Maxfield et al (1975).

9. Clayton et al (1963), Wiles & Narcisse (1971), Kennedy & Sherman (1986), and Ungar, et al (1976).

10. Tolot, et al (1958), Potter (1974), and Potter (1973).

11. Tolot, et al (1968), Potter (1973).

12. Chivers (1978), Tolot, et al (1958), and Lyle, et al (1979).

13. Potter (1973).

14. Santodonato (1985).

15. Chen (1986).

16. Ducatman et al (1986).

17. With the reports of testicular cancer, it is ironic that the ACLU has been pursuing a case against DuPont for its exclusion of female employees from DMF production lines.

CHAPTER FOUR: THE STRIKE AND THE GOVERNMENT RESPONSE

By the end of January the conflict between the needs of workers fired or unable to work and those still on the job had come to a head. Beth Lewis, the clinic social worker, was exasperated with unsuccessful attempts to get groceries, clothes, and benefits for the workers. While the company was installing the requested engineering controls, they were also contesting the workers' compensation claims. They had even gone so far as to hire the director of occupational health at Yale University, Deborah McGregor MD MPH (no affiliation to the Yale Occupational Medicine Program) as a consultant.[1] She proceeded to dispute the clinic's findings, claiming that the outbreak was due to alcohol and tylenol abuse. Uretek was not the paragon of cooperation despite their claims of such to the media. Ms Lewis also recognized that there were more issues to resolve than just those involving health and safety. Workers had no benefits, were required to work 12-hour shifts, and were fired indiscriminately. They needed more help than the clinic or an attorney could provide. The program staff agreed that Lewis should seek contacts in "the Hispanic community." It happened that Daniel Perez had recently arrived in New Haven as an organizer for the International Ladies Garment Workers Union (ILGWU) and Lewis' inquiries quickly led her to Perez.

Within 72 hours an overwhelming majority of Uretek workers authorized the ILGWU to represent them in what was to be the prelude to a bitter four-month strike. Perez called on OSHA, the media, and the mayor. He obtained benefits for the workers and demonstrated an tireless perspicacity that even Uretek's owners ultimately came to respect.

The Big Story

The Uretek story hit local papers and television on February 19, 1987. A week later a report of the medical details appeared in the Center of Disease Control's Morbidity and Mortality weekly report. In the weeks that followed, the story was picked up by the Boston Globe, the New York Times, and other major papers. Reporters set about finding out who knew what, and when. It wasn't long before documents were uncovered which indicated that Uretek had been investigated by both state and federal inspectors years before the outbreak.

The OSHA inspection, which covered only safety regulations and not health rules, occurred in February of 1979 and found 11 safety violations. The safety inspector, sufficiently concerned about toxic health risks, recommended a separate health inspection and requested a list of chemicals from company vice-president and chief chemist, John Andrews, despite the fact that he had been sent to examine safety

compliance only. Andrews responded with a letter listing eight coating names, but stating that "the exact chemical composition of the coatings is unknown to us, as the suppliers consider the coatings as proprietary".[2] Andrews stated that he was assured by manufacturers that the coatings did not contain any "unduly hazardous or toxic chemicals that would affect the safety of the people working in the plant".[3] OSHA supervisors, evidently satisfied, made no further requests and, until the outbreak, a health inspection was never performed.

The state, as it turned out, virtually stumbled into Uretek. Inspectors from the state Department of Environmental Protection inspected the Chem-Tech Rubber Company in May of 1984 for hazardous waste storage compliance. Chem-Tech occupies the ground floor above the Uretek basement operation and is owned by the same individual, Harold Hoder. One inspector, Peter Zack, was so overwhelmed by the chemical odors that he noted in his report that anyone from DEP "should have respirators to ensure [the inspectors's] safety and health". He also contacted DEP's air compliance division, recommending that they investigate possible air pollution problems outside the plant. No one ever followed up on the suggestion.

Zack did, however, document that Uretek's storage yard was full of leaking drums of Toluene, Methyl Ethyl Ketone, and Dimethyl Formamide. He later substantiated that the

company improperly disposed of hundreds of gallons of toxic waste by selling it to a fuel blending company, This fact, combined with the deceptions and uncooperativeness of Uretek management, led Zack, in November of 1984, to refer the case to the Attorney General's office.

While this case was being argued, the state DEP air compliance division finally found Uretek because of complaints by neighborhood residents of strong odors. They discovered, as already mentioned, that Uretek was emitting pollutants at a remarkable rate. Gary Dorsey describes the situation at that point:

> Negotiations were held, compliance orders served. But change was slow. The bureaucracy worked over Uretek according to traditional labyrinthine scheduling. Seventeen months later, when the occupational medicine program brought Uretek to a standstill, the state was still dickering with the company about its air pollution and hazardous waste problems.[4]

The Government Response

From the revelations of the preceding section, it is perhaps no surprise that a number of state and federal officials found themselves in the hot seat. Although the revelations of state inspectors' concerns were embarrassing, Uretek primarily served as an opportunity for state officials to score political points, as the state had no jurisdiction over health matters in the plant. State Attorney Joseph Lieberman was an early critic and lost no

time in emphasizing his pursuit of Uretek in court since 1984 for hazardous waste violations. The mayor of New Haven, Biagio DiLieto, attempted unsuccessfully to act as a mediator in the strike.

Political pressure at the state level focused on state Superior Court Justice Thomas P. Maino, who had delayed reaching a verdict in the original hazardous waste case for more than two years. What could have been a quiet back-page decision became the object of close scrutiny by the public, by other factory owners and by politicians. Uretek faced possible fines of $50,000 per day for 100 days of violations from May 15 to August 22, 1984, which totalled $5,000,000. Vice-president Andrews faced a personal fine of $5,000,000 and a 5-year prison term. On April 14 the judge found Uretek guilty and levied fines of $225,000 and $5,000 fines against Uretek and Andrews, respectively.[5] There was no prison sentence. Uretek became the first company in the state convicted of hazardous waste violations in a criminal court.[6] Although Judge Maino's decision was criticized by some as lenient, there is some belief that the penalty would have been smaller had it not been for the hepatitis outbreak.[7] The decision set an important precedent in the state for criminal penalties in toxic waste cases.

Federal Hot Potato

If there was discomfort at the state level, there was near panic in federal circles. U.S. Senator Lowell P. Weicker hand-delivered a letter to Labor Secretary Brock in March complaining about OSHA's 10-week inspection backlog. The New England OSHA director, John B. Miles, Jr., who had jurisdiction over Connecticut, had assumed his post two weeks before the Uretek story hit the press in mid-February. Unknown at the time was the fact that Miles had previously been director of the prestigious Manhattan OSHA office. He had been "reassigned" in March of 1986 after an internal probe of the Pymm Thermometer Factory outbreak of mercury poisoning, in which several workers died (of unsubstantiated causes), and at least forty others were seriously affected.[8] It was Miles' bad luck to be transferred into an explosive situation, but he responded with unusual vigor, requesting "loaner" inspectors from OSHA's Emergency Health Response Team in Salt Lake City, and calling on Dr. Ralph Yodaiken, one of two OSHA staff physicians, to review the workers' health records. The latter is extremely unusual for OSHA, which has no provision for involvement of medical personnel in local work. On July 28, 1987, six months after they were notified, OSHA cited Uretek for 34 willful and 145 serious violations of the OSH Act.[9] Fines totalling $480,000 were proposed, including $10,000 each for the 34 willful violations and $1,000 each for the 136 serious

violations, all the maximum allowable by law. Given that the average proposed OSHA penalty is less than $100, and less than $900 for willful violations, the proposed Uretek fines were notably harsh.[10] They came at a time when OSHA was increasingly under fire for alleged inactivity. Of note, both the company and its new ally after the strike, the union, contested the proposed fines.[11]

Endnotes

1. This turned out to be a brilliant maneuver as the public did not grasp the difference between McGregor's and Cullen's affiliations with Yale and their views were portrayed as an intradepartmental feud in initial reports.

2. Letter to OSHA quoted in McGraw & Kauffman, April 10, 1987.

3. Ibid.

4. Dorsey, 1987.

5. These fines are currently under appeal.

6. As of this writing, there are three outstanding civil cases involving hazardous waste from 1984, 1986, and 1987.

7. See Povinelli (1987).

8. Miles' replacement, acting OSHA director Patrick Tyson, expressing sentiments familiar in the Uretek case, commented: "How the hell could something like this happen? How could we have gone this long without the system catching it?...It is clear to me we made some mistakes". Pymm officials were eventually convicted of criminal charges. See Hirschfield, 1987.

9. A willful violation is defined by OSHA as one where a substantial probability exists that death or serious physical harm could result, and the employer knew, or should have known, of the hazard.

10. Figures are derived from Table 2 in the Report of the President to the Congress, 1985, 42.

11. Like the state fines, the Federal penalties are still under appeal.

CHAPTER FIVE: THE HISTORY FEDERAL STATE AND PRIVATE REGULATION OF OCCUPATIONAL SAFETY AND HEALTH

At first glance the term occupational safety and health seems to be a single concept. However, ensuring health in the workplace means prevention of disease; workplace safety involves prevention of accidents and resulting injuries. There are several important implications of the disease/injury distinction. The first, and most important, involves the thorny issue of causation. Normally, the causes of injuries are temporally proximate and established with ease in comparison with disease, especially occupational disease. In the case of the latter, causal events are often incremental, diffused over months or years, or involve threshold exposures. Results sometimes do not manifest for years or decades, and other causal influences clog the intervening time. (Disease is a result of a complex interplay between internal [host] and external [environment]). It follows, then, that the study of occupational disease is the characterization of disease clusters, and the methodology is primarily that of epidemiology. The study of injury, on the other hand, is the characterization of random events which occur with a certain probability, established primarily by statistical methods.[1] While the study of occupational injury has included the "injury-prone worker" as well as the

dangerous workplace, the host/environment interplay is much more complex.

Historically, the distinction is important, as we shall see, because of the difficulty institutions originally designed to mitigate the consequences of accidents have had accommodating to occupational disease. In the case of worker's compensation, the problem has been one of a round peg in a square hole. The result is that very few workers are receiving compensation for work-related diseases. The same difficulty can be seen with OSHA. While it has the word "health" in its name, it was never designed to detect or prevent health problems:

> OSHA's role, then and now, is to make rules about how work should be done safely and to enforce them, much like police. OSHA is not equipped to assess whether someone is hurt or sick, but whether rules are broken.[2]

This theme will recur as the historical roots of occupational health in Connecticut are traced in this chapter: federal and state laws, and the influence of the early voluntary groups on these institutions. A further treatment of the implications of the accident/disease in prevention and identification of workplace problems is included in chapter six.

Unhappily, the history of sick and injured workers is long, back to the beginning of recorded history. Throughout this long history, attention has primarily been directed to industrial accidents, not to workplace disease.[3] It is

curious the explosion of medical knowledge in the last thirty years, and its application in many new arenas has not led to a renaissance of occupational health. While a veritable mountain of medical writing has appeared on tobacco, alcohol, diet, exercise and their influence on health, not to mention literature on a plethora of new diseases, most of the profession has been strangely silent about occupational illness. Perhaps, as some have asserted this is due to a decline in the real incidence of industrial disease? While deaths on the job have certainly decreased since 1900, this seems less certain for non-fatal diseases. Even with the shift in the 1980's to a "service compounds and processes has vastly increased the number and severity of exposure to hazards.[4] It will be argued in the next chapter that the lack of medical literature on workplace disease reflects a failure to recognize it when it occurs, and that this in turn is due to a complex set of factors, not all of them medical.

Perhaps the media's conclusion blaming the outbreak primarily on irresponsible owners, dangerous chemicals, and a weakened OSHA is sufficient to partly answer the question why such an outbreak occurred. A complete answer though, one that provides meaningful information for the future, benefits from a look at institutions which existed in the past and how they functioned. The goal is an analysis which recognizes that events do not occur in a vacuum but in a

context, and that individuals and their actions influence and are influenced by societal structures and institutions in a complex way.[5] For one, the nature of preventing workplace injury and disease involves economic, political, ethical, and social judgments. In addition, the particular judgments made by legislators and bureaucrats are buried in the maze of official documents or obtuse administrative manuals. The net result is a remote, inaccessible and at times daunting subject, and understandably, few attempts have been made to untangle the issues. In fact, few widely available books have been written on the history of the Federal OSH act itself, although a number of critiques of OSHA have appeared dealing primarily with the function of the act which include brief historical treatment.[6] To fill this gap in the literature is beyond the scope of this modest undertaking. The focus of this chapter will be to fill a narrower but heretofore unexamined gap: the history of occupational health in Connecticut, which by necessity will include public and private efforts on the national level.

Turning to the early 1900's we find it was a simpler time in occupational health to be sure, at least for company owners. No national health or safety legislation existed. The closest things to a federal occupational health standard were limited efforts such as the "Esch Act of 1912", which placed a prohibitive tax on white sulphur, driving it out of

use. Inhalation of white or yellow sulphur, formerly the chief constituent in matches, caused decay of the jaw and death or disfigurement.[7] Other federal health projects of the day were the Federal Workers' Compensation Act of 1908, the Mine Safety Act of 1910, and the formation of the Public and Indian Health Services in 1914. Although they certainly represented improvements, these acts had a much greater impact on injury than disease in the workplace, and even this effect was constrained to a limited number of work sites. The Compensation Act, for example, extended only to federal employees. The Mine Safety Act made stipulations about construction and ventilation, but nothing was included concerning dust masks or other health protections. The states were just beginning to promulgate legislation at the time, and the scene was dominated by voluntary organizations and medical specialists mostly under the influence of industry. Like the handful of public acts, voluntary efforts that came to pass dealt primarily with safety, that is, industrial accidents, and not with diseases arising from work.

Voluntary Efforts

NSC: Prior to 1927, most of the regulatory work influencing Connecticut had been through voluntary organizations. The first of these was the National Safety Council

(NSC), founded in 1912 by prominent industrialists as a response to the alarming rates of accidental death and disfigurement that accompanied the proliferation of powerful machinery.[8] Known today primarily for their automobile and bicycle safety campaigns, the NSC expropriated the "Safety First" slogan from United States Steel, which in 1910 had formed its own accident reduction campaign in response to negative publicity. With its slogan, the NSC took the ideological high ground from which to focus blame "on the careless worker rather than on corporate responsibility for dangerous designs and practices."[9] Despite the fact that the NSC currently publishes some of the most heavily quoted workplace morbidity and mortality data, they have sponsored little or no industrial research and have downplayed the dangers of occupational disease.[10]

ANSI: A second voluntary organization, the American National Standards Institute (ANSI), was founded in 1918 (under a different name) by a group of corporations and manufacturers to "develop industrial standards through consensus." Part of their efforts have focused on such issues as nut and bolt size compatibility while others have attempted to improve occupational safety by promulgation of standards for hazard exposures. Since 1969 these standards have been reviewed by a board including government and consumer but no labor members and with 9 of the 15 members from industry. The board decides when a standards committee

has reached "consensus" by weighting and counting the votes of the members. Unfortunately, the weighting decisions are not made public.[11] Certainly this would all seem terribly arcane had not 180 ANSI standards been adopted intact by OSHA as regulations in 1971 by authority of Sec. 6(a) of the OSH act.[12] Like the NSC, ANSI has ventured into data collection as well, by means of its "USA standard method of recording and measuring work injury experience-Z16.1." Originally developed by government statisticians but taken over by ANSI in the 1920's, the ANSI statistics were the only source of work injury rates in the US until the passage of the OSH act. Many view ANSI as seriously underestimating accident rates, while they do not even bother to collect disease data at all.

ACGIH: A third organization also had a number of its standards adopted by OSHA: the American Council of Government Industrial Hygienists (ACGIH), founded in 1938. The ACGIH was a professional organization, unlike the NSC and ANSI, and adopted standards by organizational plebiscite. The standards take the form of Threshold Limit Values (TLV's) which are eight hour time weighted averages defined as "the concentration of an airborne contaminant to which workers may be exposed repeatedly day after day, without adverse effects." They are established by extrapolation from animal toxicity studies and from reports of exposure in workers. OSHA adopted the 1967 ACGIH TLV's in 1971 and they

had previously been adopted by a number of foreign governments. In addition, TLV exposure levels have been criticized for being too high. Hollstein points out that exposures are "considered acceptable if...[they] cause physiological changes in the exposed worker but the changes are not harmful."[13] For instance, TLV's for carbon monoxide, nitrogen oxides, and sulfur dioxide are four to forty times as high as the limits set by the EPA for the air people breath when they are home from work. The number of ACGIH TLV's has almost doubled since 1967 and for many the TLV has been lowered. However, OSHA has not fundamentally revised its inherited list. As these standards were adopted unaltered, they represent one of the significant inadequacies in OSHA discussed further in chapter six.

NCCI: The final voluntary organization of note is the National Council on Compensation Insurance (NCCI). Whereas the NSC and ANSI are primarily supported by manufacturers, NCCI represents the interests of the insurance industry. Perhaps the last oligopoly untouched by anti-trust legislation, the insurance industry, and the workings of its lobby group, have yet to be documented in any detail. However, NCCI's interest in the early part of the century is fairly clear; they vigorously promoted worker's compensation laws. In particular, they hoped to avoid federal regulation and underwriting and management of the funds by the states. In regard to the latter, the insurers demonstrated some

foresight: worker's compensation was to be a profitable business.

Between 1950 and 1970 private insurers have kept an average of 40 cents of every dollar of worker's compensation premiums in contrast with 20 cents for state funds and less than 5 cents for the Federal Employees Compensation act.[14] In 1974, the nationwide premium benefit ratio was 0.53 (ie 47 cents to administration) for private insurers versus 0.72 (28 cents) for state funds.[15] Even more dramatic is the fact that five states which prohibit private compensation insurance companies operated state funds with a 0.94 benefit/premium ratio, with Ohio out front at 0.96.[16] This means that it costs Ohio a little over 4 cents to return a dollar in benefits, whereas it costs insurance companies 89 cents (47 cents of cost for 53 cents benefits returned) to do the same. Although state funds are exempt from taxes and deliver fewer services than private insurers, it appears that broker commissions and profits figure into the efficiency difference. Just how much profit is difficult to say. As already pointed out, the insurance industry has enjoyed near total freedom from independent scrutiny. It is sobering to consider, as Berman points out, that "worker's compensation benefits could be nearly doubled at no new cost to employers by eliminating the role of the private insurance companies."[17]

Worker's Compensation

Although the voluntary programs eventually had a major impact on occupational health when they were adopted as standards in 1971, worker's compensation was far and away the most important single factor in the area prior to OSHA. From the beginnings of the industrial revolution in the 1870's up until the 1900's, employers had successfully avoided liability for worker injury and illness in the US. Under common law, employers were held to the "prudent person" standard, and expected to take due consideration of employee safety, including forewarning them of unusual hazards. In spite of this "counsel of perfection", workers were rarely able to recover against their employer in court. Proof of negligence on the part of the employer is difficult under the best of circumstances today, and even more difficult at the time as a result of the total dominance of employers in the legal arena. In addition to the expense, delay and uncertainty of a suit, litigants had to contend with a number of realities of the day. Coworkers or a foreman who might be key witnesses would risk firing if they testified on the workers behalf, and even with adequate testimony, the likelihood of a decision in the plaintiffs favor was small given three "doctrines" accepted as adequate defenses, sarcastically referred to as "The Holy Trinity".[18] The first, contributory negligence, absolved employers if

the employee could be shown to have contributed to the incident, even in a minor way. Likewise, employees could not collect if a coworkers;s negligence contributed, the fellow-servant doctrine. Finally, knowledgeable assumption of risk asserted that if a hazard causing injury was inherent to the work, and if the worker could be expected to have known of it, then his or her acceptance of the job precluded negligence. These three doctrines were interpreted so liberally by the courts prior to 1900 that results favorable to the plaintiff were very unusual.[19] By 1900, however, workers began winning suits in greater numbers.[20] Progressives began publicizing the plight of injured workers and their families, or in some cases, their survivors. The best known of these was the "Pittsburgh Survey", carried out by Crystal Eastman in 1907-8 which found that employers took no financial responsibility in over half the cases.[21] Most important of all, big business cried for relief. In 1910 two large business associations, the National Civic Federation and the National Association of Manufacturers, started to lobby for state worker's compensation regulations. The reasons were simple. Although large corporations could underwrite their own insurance funds, medium and small firms could not, and for them some pooled risk scheme was needed. In addition, businessmen recognized the desirability of a system of preestablished and fixed payments, upon which they might

exercise some indirect control, over one in which workers were obtaining increasingly large jury awards that could spell catastrophe for many companies. Just how this indirect control was achieved is explored in the next section. Another motivation of commercial support was the fear that public perception of an injustice had reached proportions that might spur government to alter employer-employee relations (by invalidating the common law defenses, as occurred in a few states) and give the nascent union movement an unwanted boost.

Not surprisingly, unions opposed worker's compensation legislation for reasons that we might expect: their mistrust of government interference and their expectation of improved results in the courts. It is also no shock that their opposition had little impact on the course of public policy.

The theory behind the legislation was no-fault liability, providing prompt and adequate benefits while relieving both employers and employees of the uncertainty and expense of litigation. In actuality benefits were low, most states set maximum awards at 50% of the workers income and although an improvement over some court awards, the number of high value settlements was growing in the years preceding the legislation.[22] With the acceptance of a comp award the worker forfeited the right to a common-law suit.

The first states to pass compensation legislation, New York, Maryland, and Montana, had their statutes ruled

unconstitutional by the courts. Wisconsin, in 1911, became the first state to have a system which remained in effect, followed shortly by others including Connecticut in 1914. Twenty-four states had legislation by 1925, but it was not until 1948 that all states had such laws.[23] The early laws excluded coverage of occupational disease, some more explicitly than others. Three reasons for this have been cited. First, as compensation laws were intended to supplant common law jurisdiction, and since disease claims were denied by common law, no need was seen to include it. Second, it was thought, perhaps correctly, that disease compensation would be much more expensive and place a heavy burden on industries, such as mining, where high disease rates were recognized by management, even if they denied responsibility. It was thought that illness could be handled by health and disability insurance, which distributed the risk burden over a larger pool (and except for a few diseases such as asbestosis and lead toxicity, this remains to some extent today). Finally, in the more progressive states, the laws were written with language sufficiently expansive to allow future coverage of illness, but not so broad as to alarm legislators. Massachusetts was the first state to extend coverage in this way, followed by nine others in 1928 including Connecticut, 18 more in the 1940's and the remainder between 1951 and the present. [24]

To say that all states now have compensation for occupational disease is misleading. The compensation system was designed to provide prompt and fair payment to victims of workplace accidents, and despite some significant problems with award levels and incomplete coverage of all employees, the system has had some success. Ashford points out though that "the designers of the original statutes did not foresee the slow, silent disability caused by many of today's occupational hazards, and the problems of recognizing and understanding such occupational diseases have therefore raised critical issues for the entire workmen's (sic) compensation system."[25] We will return to this point shortly.

The compensation system is heavily flawed. Some are functional obstacles, such as "inefficiency" of private insurers, discussed in the previous section, uneven coverage of workers in general, and unequal compensation of specific diseases or injuries from state to state. More serious are the flaws stemming from internal contradictions, which in turn reflect competing goals of the compensation system. These flaws bear more careful attention.

Ashford identifies five "social" goals for the compensation system and what it should ideally accomplish: income maintenance, insurance, efficiency, broad uniform coverage, and equity. It is clear that the maintenance goal is not being met. According to data amassed by Berman in

1978 from public and private sources those receiving compensation recover only one fifth of their original income on average. Four-fifths entering the system receive only medical benefits, in part because most states require disability for eight days or more to qualify for income replacement. If all of the workers entering the compensation system in 1972 received all their lost salary, the amount paid out would have totalled $18.2 billion versus the $4 billion that was actually disbursed.[26] Why not full income replacement? Are not the worker and their family entitled to live as though the worker "had remained well and sound?"[27] A proposal has already been mentioned which would allow a doubling of benefits at no added expense. But this might compete with the goal of industrial efficiency, as construed by industry as output per dollar expended. It has long been feared that "as the proportion of wages replaced is increased, the worker will have less incentive to return to work."[28] This would contribute to lost productivity and increased costs. As Ashford points out, the conflict between efficiency and income maintenance is a real one, but one which could be subject to empirical resolution through a study of return to work in relation to comp payments. This has yet to be done. The real lesson here (as with the additional conflicts enumerated elsewhere) is that internal inconsistency in the goals of programs, such as worker's

comp, lead to political struggles in which "the relative power of private interests may determine the outcome."[29]

Perhaps the core inconsistency lies in the goal of insurance coverage. As already described, the initial intent was to insure workers against a decline in income secondary to injury and to protect industry from crippling liability judgments. But the type of insurance that was created is far better suited to the latter as it "treats the wastage of man-power as it does the wearing out of machinery."[30] It is logical of this compensation system then to allow industry to pass on the costs of compensation to the consumer much as they would depreciate their equipment. Schneider stated in 1922 that:

> The plain purpose of the compensation law is to make the risk of accident one of the industry itself, to follow from the fact of injury, and hence that compensation on account thereof should be treated as an element in the cost of production, added to the cost of the article and borne by the community in general.[31]

This fact, combined with a number of realities which insulate employers from the true costs of injury and illness, make compensation much less effective than it might be at promoting prevention. This is taken up again in Chapter six.

Returning to worker's compensation and occupational disease, it is important to recognize how uncomfortably the comp system has accommodated the inclusion of disease. In contrast to accidents, which are discrete temporal events

with clear and often dramatic causal connection to any injury, disease is often slow to develop, difficult to distinguish from nonoccupational illnesses and the consequence of multiple causal factors. A classic illustration is lung cancer, which can develop 25 to 35 years after even brief exposure and which presents essentially in the same manner as Tobacco induced lung cancers. Furthermore, it is known that cigarette smoking increases the risk of lung cancer and other lung diseases in exposed workers. The Uretek hepatitis cases are also illustrative although unusual in their short latency period. Workers frequently run up against statutes of limitations for filing compensation claims. The U.S. Department of Labor recommends a one year limit after the victim becomes aware of an illness and that the illness is work-related. In 1972 only 26 of 58 worker's compensation statutes met this standard.[32] The problem is further compounded if the worker has moved on to one or more employers or retired in the intervening years. This is the rule rather than the exception with occupational lung disease and cancer. Retired workers are particularly vulnerable, as the comp system requires the claimant to be in the workforce to be eligible for payments.

Multiple and uncertain etiologies also present problems for a system which treats disease as a special sort of injury. For one, it has left the insurance carrier with an easily abused basis for contesting claims as it is quite

difficult to achieve the same level of certainty in assigning cause in the case of disease as opposed to injury. This situation exists because the comp law takes an overly simplistic view of causation, treating it as a question amenable to swift resolution by one or more "experts". While this might be true of injuries that result from accidents, it is decidedly unproductive in the case of disease. The philosophical roots of this approach to problems are explored further in the section on obstacles to reform, in chapter six. For the moment, the point is that multiple causation introduces an element of irreducible uncertainty, beyond what is encountered in a workplace accident. This makes for long and acrimonious compensation hearings (eg the Uretek hearings still in progress a full year after the outbreak), or worse, workers being denied compensation.

Although the response to workplace disease is not the central issue of this thesis, it is important to understand the very complex system that is worker's compensation. In fact, it is impossible to analyze prevention and recognition without such an understanding. By looking at the development of worker's comp legislation historically certain patterns become clear. In many states Government has abdicated its role, and in so doing, placed a heavy burden for injury and illness on workers and their families. A nation-wide study of 2.2 million workers disabled for at

least three months found only 24.7% ever receive compensation. 68.5% never even bothered to apply and this appears to be because they did not expect to get anything. Less than 1% of all compensation payments before OSHA were for occupational disease, and four years after the OSH act, the number was 1.35%.[33] All of this suggests a huge reservoir of uncompensated victims. Why is this so? The reasons are similar to those for the delay in recognition of the Uretek cases. Workers who got sick were fired and not eligible for compensation. Moreover, the cause and effect relationship of work and illness are clear neither to the employee nor the physician. As with Uretek, it can be a long time, and many sick workers, before this concordance is established. Particularly worrisome in this regard is the vast and rapidly increasing stock of chemicals to which workers are exposed, many incompletely tested for toxicity, and the time lag for appearance of information about their hazards.

Perhaps the clearest, and unfortunately the most familiar pattern, is the ability of large companies to set societal priorities when legislation fails to resolve conflicting interests of workers and business. The next section will examine the development of occupational surveillance in Connecticut from the same historical perspective.

State Health & Safety Programs

For a number of reasons, most of the early activities in occupational health occurred at the state level. For one, the response to workplace tragedies was most intense on a local level. The most famous of these disasters was the Triangle shirt-waist factory fire of 1911 in which 146 women died in a Greenwich Village factory. Many burned to death, unable to escape through blocked exits, others jumped eight stories to their death rather than be incinerated. It is worth noting that some recognition had been brought to working conditions in the garment industry just the year before the fire by a massive strike. Labor activism of this type was another source of pressure on the local level, but not nationally, as the unions were insufficiently powerful to generate federal activity in the 1920's. Even local influence was restricted to areas like New York with its relatively strong unions. In the case of the garment strike, the result was a city sanitary authority, which in any event did not avert the Triangle disaster. In a pattern which repeats itself throughout the history of workplace disasters, a committee was formed after the Triangle fire "to meet the public protest", in the words of New York State Senator Alfred Smith.[34]

In reviewing the historical record for significant disasters and outbreaks, two important facts emerge. First, far fewer accidents have become part of the historical

record than one might expect given the documented conditions in factories of the day. In the case of the Triangle fire, a UPI reporter happened to be strolling through Washington Square as the flames ignited and proceeded to phone live reports to the telegraph office as the tragedy unfolded. Few accidents benefit from such exposure, especially smaller, less dramatic ones that occur far from Midtown Manhattan. Furthermore, diseases lack entirely this sort of immediacy, upon which the media thrives. A case in point is the Gauley Bridge, West Virginia outbreak of the early 1930's, in which more than 700 tunnel miners died of acute silicosis, dwarfing the better known mining and factory accidents of the era and yet almost completely unknown today, even among labor historians. There are a number of reasons why Gauley Bridge may have fallen into obscurity, including a concerted effort to destroy records by the company, Union Carbide. It does at least raise the question, however, of how the media and scholars react to an epidemic of workplace disease as opposed to an accident. Certainly, an outbreak of a newly recognized disease, as was the case at Gauley Bridge and at Uretek, offers greater "plausible deniability" for an employer than a fire or accident. In the Gauley Bridge case the employer, Union Carbide, virtually escaped both public censure and significant financial responsibility.[35] The Triangle fire is unusual in that it did have an indirect effect on other

states, perhaps because the drama and immediacy captured the media's attention.

The Connecticut Experience

Within two years of the Triangle fire three states, New York, Connecticut and Ohio, organized programs in factory inspection. Massachusetts had an early lead in this area with "power of closure" authority for grave hazards accorded to factory inspectors in 1852.[36] By 1890, 14 states had statutes providing authority for factory and workshop inspectors. In most cases, however, these laws were window dressing as inspectors were political appointees and unlikely to ruffle business feathers. Few were trained to recognize safety hazards, and expertise in disease prevention was almost non-existent.

The history in Connecticut was typical. Factory inspectors were appointed by the Governor, with consent of the Senate. Their responsibilities were as follows:

> He shall examine all buildings where machinery is used and report to the Governor as respects safety to life and health of such places. Shall see that factories are properly lighted, ventilated and clean. Shall have the power to require provisions (sic) of toilet rooms with the state Board of Health. Shall cause excessive dust to be removed. Shall inspect houses where certain forms of work are conducted and shall order them to be kept clean and sanitary condition, well lighted and ventilated.[37]

The authority to require restrooms is a bit more circumscribed than the power of closure enjoyed by Massachusetts inspectors. Moreover, responsibility for factory conditions was divided among factory inspectors, local and county health officers, the tuberculosis commission and the bureau of labor statistics.[38] Though data on disease and accident rates in Connecticut at the time is not available, a newspaper story stated that accidental deaths dropped by 4000 per year from 1915 to 1922 suggesting a far from trivial rate of mortality and presumably morbidity.[39] This situation obtained until 1922 and the arrival of a new commissioner of Health from Massachusetts, Dr Stanley H. Osborn. Dr Osborn, a graduate of Tufts Medical School in 1914, had served for three years as state epidemiologist before his appointment as Deputy Commissioner of Health in 1920. Promoted to commissioner in 1922, he lost little time in expanding the role of the department of health in industrial hygiene. Up to that point the department's role in workplace protection was primarily imaginary. Organized in 1878, the state board could make investigations and issue reports on almost any subject affecting public health and safety but had no authority to remedy any condition discovered by its investigations.[40] Recognizing that incidence data was required to direct investigations, Osborn had the reporting statute of 1921 revised to allow reports of workplace disease to come to the department of health

instead of to the commissioner of factory inspection.[41] The statute already required any physician or patient who believed they were treating or suffering from a disease "contracted as a result of the nature of...employment" to contact the department of health within 48 hours. Just how often the law was observed, and by whom is not known though it did provide both for a fee to Physicians (50 cents) and a fine (10 dollars) for failure to report. Writing in 1928, the physician who ultimately took responsibility for occupational health in Connecticut commented:

> No appropriation was provided at that time, however, with which to secure the observation of this statute, so that like another I need not mention, it was honored more by its breach than by its observance and occupational disease reports were fragmentary to say the least.[42]

Irrespective of the actual number, the reports made pursuant to this law served Dr. Osborn when he returned to the legislature in 1927 to request a full-time physician for the study of occupational disease. Osborn was successful in persuading the legislature to appropriate funds to "investigate and make recommendations for the elimination or prevention of occupational diseases". The debate which occurred about the bill is interesting if only for its similarity to some of the statements made during the Uretek incident.

In the discussion, a legislator asserted that "98% of the accidents are due to the carelessness of the operator." Osborn responded appropriately that it depended on the type

of injury: "Anthra (sic) is not due to the carelessness of men."[43] Osborn goes on to say, "we are not asking money to treat disease. What we are asking is money for the prevention of disease." A month later, having persuaded the Health and Safety Committee, Dr. Osborn encountered stiffer resistance amongst Appropriations committee members. Republican Senator Edward F. Hall maintained that all occupational health problems were those of "proper machinery..and the personal habits and home conditions of the worker."[44] Consistent with this view, Senator Hall opposed the appropriation on the grounds that "the employees should solve [it] themselves."[45] The views of the Senator are not surprising given that many were factory owners, nor were they particularly original. What is notable about the session is the articulate and impassioned defense of the proposal by two representatives of the Connecticut Manufacturers' Association. Making a point which is still to be fully appreciated, a Mr Walsh stated that:

> all disease is an economic loss to the people concerned and to the state. The state through its sanatoria is today taking care of a good many cases probably of occupational diseases. It is possible by making a careful study of disease to very much limit it. As you all know that is the way we have eliminated diseases.[46]

His colleague Mr Smith added that "if $17,500 did nothing but solve that one condition in Danbury, I think it would more than pay for itself." Given the experience of the Yale clinic, and statements in the press by industry, these would

be enlightened views on their part even by contemporary standards. They are certainly atypical for the time and raise the issue of whether they owe more to personal allegiance to Dr Osborn than to forward thinking. There is unfortunately no record of how the manufacturers' association arrived at its position. In the annual report of the association for 1927 there is a suggestion that concerns about limiting compensation claims played a part.

> While the health department is not concerned in the worker's compensation aspect of this question, one effect of the work contemplated by the department will doubtless be a diminution of occupational disease claims[47]

If indeed this was the principal reason for their support it is a curious one. It was perhaps not evident in those early years of worker's compensation that insurance premiums would not closely follow the history of claims at a particular plant. Although other influences may have played a role in this case, it may represent anecdotal evidence that premiums truly tied to costs might induce employers to take an interest in preventive measures. Just such a proposal is included in the final chapter. At any rate, the proposal passed over the objections of several legislators, and the sum of $17,500 dollars was appropriated.

Dr Osborn used the funds to hire Dr Alfred S. Gray as director of the newly created division of occupational diseases. It was the first state to create an agency charged solely with the elucidation of workplace disease and

it went far beyond the "safety first" atmosphere of the day. It was, however, consistent with the philosophy of employer beneficence prevalent at the time. The approach of the department was that of cooperation with industry, with the assumption that once brought to its attention, industries would correct hazards as a matter of "good business". They had the power to inspect and recommend but there were no provisions for enforcement.

> The department is empowered to make investigations of any condition reported to it or suspected of causing occupational disease, and the result of its investigation cannot be used in court compensation cases.[48]

There is evidence that in cases of imminent threat to life the health department could shut down a workplace, but this occurred rarely if ever.[49]

The work of the department rapidly expanded, and there is evidence that it was effective even without punitive powers. In 1928 an engineer trained in industrial hygiene and laboratory technique was added and a laboratory was opened. The first year there were a "dozen or two" inquiries. By 1935 the number had grown to 463 requests for information, thirty-two technical studies and 148 site visits. According to a report by Dr Gray, the work of the department "resulted in definite improvement in working conditions, changes in processes, substitution of materials, and, in a number of instances, the purchase of entirely new

equipment, not as the result of mandatory orders but because of the fact that these industries were given definite concrete information on the effects of the environment on health."[50] Major studies included mercurialism in the hat industry and lead toxicity. The hat industry study led in 1941 to a ban on the use of Mercury to "cure" fur felt, particularly important as 50% of the hat industry was based in Connecticut. Many other states followed Connecticut's lead with a mercury ban of their own.

As it did throughout the U.S., the Second World War expanded interest and all important funding to occupational health in order to achieve peak production efficiency. The bureau of industrial hygiene, as it was then called, expanded to 28 employees including physicians, nurses, chemists, and industrial hygienists/engineers.[51] This may have been the largest industrial disease operation in the country. Prior to the war, Connecticut already had the largest staff and budget per number of industrial workers of any state.[52] It was undoubtedly this unparalleled level of funding which allowed the bureau to pursue vigorous workplace surveillance. A 1940 annual report tallies 25 studies of potential hazards, 367 site visits, investigation of 455 cases of disease and the receipt of 1066 requests for information.

There were several unique aspects to the work of the bureau at that time, in addition to its size and high level

of activity. Most significant was the presence of physicians who carried out real and informed evaluations of workplace health. In the words of Dr Osborn, the duty of the physician was "to appraise the health of the workers in terms of exposure to toxic substances...and to assist in the early recognition and the prevention of occupational disease.[53] It is precisely this type of surveillance that is entirely lacking in the current system and is one of the prime weaknesses which allowed the Uretek outbreak to occur.

The other significant activity of the bureau in the postwar period was the regular inspection of Connecticut workplaces. Unlike the current system whereby OSHA only inspects by management or labor request, a team including a physician might inspect on the basis of reports of occupational disease gleaned from compensation records, company health department files, and state department of labor inspections. In addition,

> routine industrial hygiene surveys [were] conducted periodically by chemists or engineers...The interval between surveys determined by the hazards associated with the manufacturing processes. New plants, or changes in techniques and processes of old plants, require[d] immediate investigation.[54]

This almost certainly suggests a level of surveillance of industry in the state which has not been matched to date. Dr Wistar Meigs, former director of the Yale Institute of Occupational Medicine (1947-70)[55], recalls that the state bureau was managing to visit registered workplaces at least

every two years. Dr Osborn did not note whether these visits were surprise or announced, nor for how long they persisted. He did record a large variety of investigations including airborne and biological monitoring (urine) for lead in brass, battery, and metallurgical industries, chlorinated hydrocarbons in degreasing and solvent reclamation operations, toluol in rubber manufacture and spray painting. A study of Para-Nitro-Phenol measurement in urine and blood cholinesterase activity in workers exposed to parathion, an insecticide, is sufficiently sophisticated to have been taken from the pages of a 1980's journal. What perhaps had begun as a naive but well-meaning agency had become a very sophisticated occupational health program.

Boundaries were drawn quite broadly on the bureau's scope. Dr Osborn wrote of the "necessity of considering mental health as part of an occupational disease program" and cites poor housing and recreation facilities, repetitious and routine work, and dependence on others for economic security as reasons for this need. He placed a priority on abatement of noise and heat and hired an audiometrician for screening purposes. He promoted the distribution of educational material on family and community health including brochures on cancer, nutrition, women in industry, child care, venereal disease, tuberculosis, and mental hygiene. A clinic was established to evaluate workers with abnormal heart rhythms and the department

contemplated providing a dentist to visit plants. Dr Osborn persuaded eight small Hartford factories to pool resources and hire a full-time experienced industrial physician. According to Dr Osborn, the plan in its sixth year of operation in 1952 "has worked out to the satisfaction of labor and management and has paid for itself in savings."[56] To this roseate view of the department, and acknowledging the radical nature of the approach just detailed, it is crucial to add that no objective data exists as to how well the bureau detected workplace hazards and whether they were appropriately abated. A UAW official complained at 1973 hearings that no fines or legal action were ever instituted.[57] However, at least within the industrial hygiene field, the state's program was well-respected. Dr Osborn chaired the industrial hygiene committee of the Conference of State Health Authorities of 1928 to at least 1940, and the state's chief industrial hygienist chaired the American Conference of Hygienists for a number of years.

The decline of the program in the 1950's suggests that not everyone appreciated their efforts. As is sometimes the case, the decline is less well documented than the ascendancy. Dr Meigs recalls that Dr Gray retired in the 1950's and that Ken Marckasson, an industrial engineer who replaced him, was much less persuasive with the legislature, perhaps because he was not an MD. Dr Osborn retired as Commissioner of Health in 1959 after 37 years, earning him the title of

"dean" of health commissioners.[58] Almost certainly the growth of the state department of industrial hygiene was due to the duration and intensity of commitment and the respect he commanded among legislators.

Even before Osborn's retirement, the department had begun to shrink slowly from attrition, a trend justified by legislators on grounds that they had done such a thorough job, occupational disease was rapidly waning. According to John Geil, appointed chief industrial hygienist in 1967, the department had dwindled at that point to five industrial hygienists and a ventilation engineer under the direction of Joseph Staper, a physician.

The history of occupational health in Connecticut is of a cyclic waxing and waning of attention and effort, while almost every other area of equivalent importance in medicine experienced exponential growth. The first surge of activity in the 1920's is followed by a levelling off in the 1930's, and then an explosion of activity during and immediately after World War II, again followed by a diminution of effort in the late 1950's and 1960's, this time more pronounced than in the 30's. Certainly the end of the New Deal and the crest of anti-labor sentiment accompanying McCarthyism played a role in the late 50's in decreasing the popularity of occupational health. Curiously, the prosperity and social unrest in the 1960's did not translate into improved worker health, at least not quickly, perhaps because of a

dilution of energy that accompanied the push for other health legislation such as Medicare and Medicaid. While the passage of the OSHA act in 1970 clearly presaged vast improvement in occupational health in most states, it can be seen as another phase in the cycle of oscillations, and, at least in Connecticut, not necessarily the peak of an upward spiral of progress when compared with the postwar bureau.

OSHA & the State: By Whom? For Whom?

The changes in state occupational health accompanying OSHA are confusing but not terribly important in the end. The decline of the bureau of industrial hygiene had occurred years before and although with a staff of six it may have been more effective than OSHA is today, it was hardly the groundbreaking effort it had been at its peak. After a brief experiment with a true focus on occupational health and prevention, the pendulum had again returned to the safety quarter.

After years of floundering under the fragmentary authority of the Walsh-Healey Act which established standards for and authorized inspection of government contractors, the Federal Government finally promulgated national legislation in 1970. The process had begun officially in 1966 at the behest of President Johnson. The precise historical factors which motivated the president

have not been extensively studied to date, and such an undertaking is beyond the scope of this paper. Certainly, the environmental movement's politicization of the effects of pollution and the concerns it raised about chemicals did much to break the trail and focus concern on the workplace. In any event, the departments of Labor and HEW both began development of a federal program and quickly became ensnarled in a 2-year bureaucratic tug-of-war over who would ultimately be in control of the new agency. Bold maneuvering by the Secretary of Labor persuaded the Bureau of Budget and the president to accept the labor proposal and Johnson called on Congress to pass enabling legislation in 1968. The legislation stalled, due in part to vigorous opposition by business in the form of the Chamber of Commerce. As we have seen before OSHA, a disaster and the outrage which ensues is often required to overcome opposition to occupational health legislation. In 1968, 76 workers died in an explosion at the Farmington Mine in West Virginia. Congress responded in 1969 with the Mine Safety Act, and in 1970, answering the hue and cry of both public and labor, the Occupational Safety and Health Act. A further analysis of OSHA beyond that here and in Chapters 2 & 3 is, unfortunately, not within the limits of this project.[59]

For the sake of completeness, the events in the state in the early 1970's as a consequence of the OSH Act will be briefly discussed. The federal OSHA legislation passed in

1970 encouraged states to develop their own programs, provided they were at least as stringent as the federal act, and, as an inducement, offered to provide 50% of the annual costs. At that time responsibility was divided in Connecticut, with the Health Department responsible for workplace health, and the Department of Labor, and its inspectors, looking after safety. From 1971 to 1973, Federal OSHA existed side by side with the aforementioned state structures and developed a reputation for action, at least comparatively. Connecticut was one of the first states to file with the federal government for approval of a state plan and in February 1973, public hearings were held for comment on the proposal. The plan had been developed by the Department of Labor without input from the Department of Health or Dr Staper. By and large the opposing testimony was very well thought out and researched. The opposition included organized labor, citizens' groups, and students and faculty of Yale law and medical schools. Many of the most incisive critiques cut to the flaws of federal OSHA as most of the state plan was lifted verbatim from the federal statute. However, the principal concern of many opponents was that state supervision, especially by the Department of Health, would be inadequate. Mr Michael Kane, representing the now defunct New Haven Citizens' Action Committee, stated:

The testimony and experience of the Unions that we

> have been in touch with have shown the inadequacy of state enforcement, particularly by the Department of Health. As an example, in 18 years the Connecticut Health Department...has referred exactly one case...to the state Attorney General's office for possible prosecution...In contrast, the Federal OSHA office for this region, although it is understaffed, has compiled a relatively impressive record of protection of worker health within the limits of federal law.[60]

Kane also pointed out that in 1968 Connecticut fielded 37 fish-and-game wardens (in a state not renowned for its hunting and fishing), and the same number of state workplace inspectors. Kane's views were echoed by representatives of Labor at the meeting. With two OSHA inspectors covering the state in 1988, a return to even the 1968 level, viewed by Kane as woefully inadequate, would be welcome. Kane went on to say that much of the mistrust of the state centered on a feeling that they would back away from large employers who were fighting OSHA in court at the time (e.g. United Technologies). The decline of OSHA under President Reagan could not, of course, be foreseen in 1973, but it is ironic that in the wake of Uretek, Connecticut unions have called for a return to state authority.

Another set of concerns about the state plan, especially relevant to the Uretek case, was raised by the Yale Lung Research Center. Speaking for the group, Janet Schoenberg, a public health student, described three major areas of concern about the state plan that apply equally to federal OSHA. Noting that much emphasis had been placed on safety problems in industry, Schoenberg highlighted that the

"magnitude of industrial health problems is grossly underestimated".[61] The group recommended that safety inspectors be trained in rudimentary hygiene technique so that they might notice health hazards. Certainly this would be a welcome addition to federal OSHA, as well as possibly averting the necessity of separate health and safety investigations that led to the ignored safety report on Uretek in February 1979, and the needless continuation of the hazard for seven additional years. Schoenberg also took issue with the state's proposed five-year inspection interval, describing it as "totally inadequate" given the seriousness of certain health hazards and the frequency of changes in industrial processes.[62] Again, it is difficult to find words to describe the degree to which workers are at risk in the current system, with inspections much less frequent than every five years on average. The final recommendation of interest was that medical surveillance be an explicit part of the act. As we have discussed, this is one of the major failings of the OSH Act as well and a recommendation which will be reiterated in Chapter six. The other testimony against the state plan had to do with promulgation of standards, composition of the review commission, and other points more germane to a discussion of the federal OSH Act. The testimony in support of the state plan came from the Manufacturers' Association, the Business and Industry Association, and small factory owners, all of

it resting on the basic premise that state and local supervision is better than federal oversight. One final point concerning the hearing worth noting is the prophetic testimony of Leonard Dube of the Connecticut UAW. Mr Dube made the following statement to the state panel:

> Between the citation appeal system, the possibility of long abatement periods, and the variance system, the clever employer can keep a modern day sweat shop humming for literally years before he feels the minor pinch of an insignificant civil fine, or, at the worst, a suspended criminal sentence.[63]

This is basically a description of the way Uretek conducted itself in regard to its DEP and its OSHA violations, and as some would say, with the predicted outcome. In spite of the many flaws identified in the state bill, the Labor committee reported it favorably to the General Assembly.

The bill went rather rapidly to the House in April, and in May to the Senate, where it was passed. There was a considerable amount of acrimonious debate, some of it reflecting insufficient time to consider such a complex piece of legislation. Those in favor of the bill, primarily Republicans who held majorities in both chambers at the time, argued that it provided for two groups of employees not covered by the federal act - those of municipalities and the state, which was true. Another argument offered in favor was that the state plan would lighten the onerous burden of federal stipulations on Connecticut industry and avert imminent shutdowns and layoffs. The hyperbole

involved in this position was evident to the opposition at the time, and certainly to any contemporary reader, but the Democrats did not succeed in defeating or amending the bill. A final point, and perhaps the most important, is that supporters estimated that the state would receive $350,000 during the first year of implementation. So the act was seen as a way to obtain federal funding for a state agency with the sweetener that, if necessary, they could double the size of the department at no cost to the state (there were 45 state inspectors at the time, including only 7 qualified to inspect for workplace health violations, and 8 OSHA staff members for the region). It was the prospect of a large grant from Washington toward a program for which Connecticut was already paying that ultimately swayed the legislators.

In 1977, after four years of experience with the state-administered program, the General Assembly reversed itself and returned full authority to the federal government, except for state and municipal employees who are not covered by the OSHA legislation. Complaints with the state plan were of duplication, of poor quality of inspections, and, most of all, of unnecessary expenditure. Suddenly the state's 50% share, some $311,000, seemed too much to pay with a tightened fiscal climate, and opponents of the original bill were able to reverse the 1973 action. Not surprisingly, the state agency with its hobbled medical branch had dealt poorly with health hazards and had been

embarrassed by surprise OSHA visits to plants such as Groton's Electric Boat shipyard, which uncovered willful health violations. A federal review of state performance showed that 96.3 % of inspections were in the safety area and only 3.7% in the health area in one six month period. Even former supporters of the state plan recanted. As one legislator put it:

> Connecticut OSHA can respond well within a very limited area of capability, maybe retail stores, maybe beyond that, but in terms of the industrial sector, they have not been doing the job. And I say this as someone who voted for putting OSHA under Connecticut in 1973.[64]

Though the better informed individuals were skeptical of federal OSHA's abilities as well, there was a climate of renewed optimism with President Carter's appointment of Eula Bingham as OSHA's head. As Uretek has so amply proven, the promise of decent workplace conditions made by the OSH Act is yet to be realized. The next chapter will look at obstacles to creating a healthy workplace highlighted by the Uretek case and how they might be surmounted.

Endnotes

1. There is, of course, an overlap of methodologies. Epidemiological methods are applied to injury and probability to disease. Disease is, however, more idiosyncratic and host-dependent.

2. Cullen (1987), B5.

3. There are notable exceptions. See, for example, Paracelcus (16th century Europe) on diseases of miners and metal workers; Agricola (16th century) also on mining and metallurgy; Bernardino Ramazzini (1633-1714) is acknowledged as the parent of modern occupational medicine. His treatise on hazards in the medieval crafts, De Morbis Artificum reads well even today.

4. It would be satisfying at this point to quote some statistics to substantiate the rise in occupational disease. The temptation to do so has gotten the better of more than a few otherwise incisive critics of the current system (e.g. Berman (1978)). This point actually takes us right to the heart of one of the central issues in occupational health today, that of a data base. The view propounded in chapter six holds that the data base is currently so flawed as to be useless. So, although numbers to support the views in the text can be found, they come from the same flawed statistical base or unreferenced estimates as the numbers used to contradict them. Unfortunately, it is not possible at this time to make definitive statements about events as fundamental as the number of deaths on the job in America. For purposes of scale, the most reliable estimate cites 100,000 annual deaths due to occupational disease and 390,000 new cases of occupational illness per year (from the Office of Technology Report (1985)). By comparison, death from AIDS in the US last year totalled approximately 13,200, with 21,917 new cases identified amongst the 1-2 million Americans exposed (from CDC AIDS survey report, January 25, 1988). Thus, nearly ten times as many people died on the job last year as died of AIDS. Keep in mind that while the AIDS data is highly reliable, the occupational figures may underestimate actual figures because of the lack of a reliable reporting system. The AIDS example, while not a true analogy for occupational disease, underscores the extent to which a problem as significant as occupational disease is unrecognized. Reform of the data collection system is clearly a high priority.

5. Sociologists and others use the term "dialectical" to describe this type of historical analysis.

6. The best and broadest of these summaries is the out-of-print OTA report (1985). See also Berman (1978), Ashford (1976), Smith (1976). The following were not available to this author: McCaffery, D.P. 1982. OSHA and the Politics of Health Regulation. New York: Plenum Press.
Mendeloff, J. 1979. Regulating Safety: an Economic and Political Analysis of Occupational Safety and Health Policy. Cambridge, MA: MIT Press.
Mintz, B. W. 1984. History, Law and Policy. Washington, DC: Bureau of National Affairs.
Nothstein, G. 1981. The Law of Occupational Safety and Health. New York: MacMillan, The Free Press.

7. See OTA report (1985), 53. Berman (1978) credits an organization called the American Association for Labor Legislation (AALL) with passage of the act but says it was "a success impossible on a broad scale "(17). Interestingly, the AALL was headed at the time by Yale Professor Henry W. Farnam, MD. See Selleck & Whittaker (1962), 43.

8. See OTA report (1985), 205 and Berman (1978), 76-8.

9. Berman (1978), 78.

10. OTA (1985), 206. The NSC data publication is called Accident Facts.

11. Ibid, 206.

12. See section on OSHA. Sec 6(a) reads in part as follows:

> ...the Secretary shall, as soon as practicable during the period beginning with the effective date of this Act and ending two years after such date, by rule promulgate as an occupational safety or health standard any national consensus standard, and any established Federal standard, unless he determines that the promulgation of such a standard would not result in improved safety or health for specifically designated employees. In the event of conflict among any such standards, the Secretary shall promulgate the standard which assures the greatest protection of the safety and health of the affected employees.

13. Quoted in Berman (1978), 81-2.

14. Ashford (1976), 389.

15. Berman (1978), 64.

16. Ibid.

17. Ibid.

18. Grillo (1964), 240.

19. Ashford (1976), 388 and OTA (1985), 207.

20. See Posner, R. (1972) "A theory of negligence" Journal of Legal Studies. V 1, January, pp 29-96 quoted in Ashford (1976), 389.

21. Levy & Wegman (1983), 11 and OTA (1985), 207.

22. Ashford (1979), 389

23. OTA (1985), 208.

24. Ibid.

25. Ashford (1979), 411

26. Berman (1978), 62.

27. Beers (1928), 592.

28. From the Report of the National committee on State workman's compensation laws, p. 56, quoted in Ashford (1976), 393.

29. Ashford (1976), 393.

30. Beers (1928), 579.

31. Schneider, Workman's Compensation Law, 1922 quoted in Beers (1928), 579.

32. Ashford (1976), 413.

33. National Commission on worker's comp study quoted in Berman (1978), 68.

34. From Smith's autobiography in Stein (1977), 198. The committee included luminaries such as Robert F. Wagner, Sr., Samuel Gompers, and Frances Perkins, who was an eyewitness and later to become Secretary of Labor through Franklin D. Roosevelt's three terms. Smith himself became a four term Governor of New York and a Democratic Presidential candidate in 1928.

35. See Cherniak (1986).

36. OTA (1985), 209.

37. Connecticut Department of Health records, unpublished.

38. Ibid.

39. **Hartford Times**, November 6, 1922.

40. Connecticut General Statutes (1878).

41. Connecticut Acts (1923).

42. Gray (1928), 6. The reference, of course is to prohibition.

43. Public Health and Safety Committee Hearing (1927). "Anthra referred either to Anthrax, an infectious disease of wool-handlers, or anthracosis, a pneumoconiosis due to coal dust.

44. Appropriations Committee (1927).

45. Ibid.

46. Ibid.

47. From Annual Report of the Industrial Relations committee, Connecticut Manufacturers association, 1927, Carl F. Dietz, Chairman (verbal communication to the author).

48. Gray (1935).

49. The Former director of the Yale institute of occupational medicine, which existed from 1947-1970, could only recall one instance in which the health department closed a plant, and it occurred during World War II. Personal communication from Wistar Meigs, MD.

50. Gray (1935).

51. Of note, the division of occupational disease was rechristened the bureau of industrial hygiene, without any change in structure, leadership, or authority sometime before 1935. It is not known if the reason for the change involved objections to the term "occupational disease", or a response to the rise in popularity of industrial hygiene.

52. Table in Conference of State and Provincial Health Authorities (1940).

53. Osborn and Johnston (1952).

54. Ibid.

55. The institute was the temporal precursor to the current program at Yale though they share little common history. The institute had been gone for nearly 10 years when Dr Cullen started the current program. For the record the Yale University Lung Research Center, a federally funded project, did carry on clinical work in the area of asbestos-related disease in the early 1970's, but did not engage in workplace visits or treat workers with non-asbestos complaints. The clinic was operated by Drs J. Bernard Gee, Arend Bouhuys, and Charles Mitchell. Unfortunately, no records from the institute survive. Throughout its existence it remained small, with no greater than two physicians (Wistar Meigs, who had been trained at the Army Industrial Hygiene lab and Ronald Bucken), a hygienist and a nurse. It carried out factory visits and clinic consultations as does the current program, though on a considerably smaller scale.

56. Osborn and Johnston (1952).

57. Information in this section is from the 1952 APHA presentation, unpublished.

58. Dr Osborn died in 1975 at the age of 84, a firm believer in prevention through education rather than compulsion. His resume shows that he lectured at Yale Medical School for the 1923 academic year.

59. See endnote #4 for references.

60. Labor & Industrial Relations Committee Hearings (1973, 98).

61. Labor & Industrial Relations, February 9, 1973, 87.

62. Ibid. 88.

63. Ibid.,72.

64. Senator Denardis, Senate debate, June 8, 1977.

CHAPTER SIX: PROGNOSES AND PRESCRIPTIONS

The drama of the Uretek story has done much to publicize the plight of workers' health in the media. However, in its tendency to simplify, and its disregard for historical context, the reporters told only part of the story. Yes, OSHA had been ravaged by budget cuts and this in part accounted for the lugubriousness and seeming disinterest OSHA displayed in the Uretek case. But the media missed the fact that OSHA has little capacity to assess workplace health. Moreover, the fact that the state Health Department had very successfully incorporated just those functions in the 1930's and 40's went completely unrecognized. As Cullen has pointed out, "uretek was neither the first, nor the worst case, of life-threatening disease stemming from the workplace.[1] Of the more than 2,000 cases of Occupational disease seen by the Yale clinic since 1979, more than a few have been fatal and most did not occur in facilities described as "Dickensian". This final chapter is a synthesis of a deeper analysis of the Uretek outbreak. It provides some subtler reasons why Uretek was missed, what is needed to prevent future Ureteks, and some larger societal obstacles which need to be overcome. Cullen remarks:

> If that awareness [of Uretek] could be transformed into a deeper understanding of what transpired and why it will continue to happen until people demand a change, perhaps something worthwhile may yet be accomplished.[2]

It is in this spirit that this chapter is proffered.

Recognizing Workplace Disease: Why Uretek Was Missed

Thus far the focus has been on the bureaucratic deficiencies of OSHA: lack of funding, lack of health enforcement, and scarcity of standards. OSHA's failings can not be blamed solely on a failure of political will, however. There are a number of characteristics of occupational illness that make it so difficult to recognize and are in part responsible for OSHA'S inadequacy.

Foremost are three aspects of occupational disease alluded to in earlier chapters. First, occupational disease is difficult to distinguish from nonoccupational disease. Rarely is there any sign, aside form the work history, which provides a clue to the etiology. The other two elements, multifactorial causation and long latency, were already mentioned in regard to the workers' compensation system. These two together make it difficult to establish the cause of workplace illnesses. In the Uretek case, liver injury was a nonspecific response to chemicals; viruses and alcohol & drug abuse had to be excluded. In the case of asbestos related lung disease, on the other hand, cigarette smoke has been shown to be a synergist with

asbestos fibers in the production of lung carcinomas and must be factored into attributions of causation. These examples are provided to show that it is often easy to dismiss a workplace disease as due to some agent outside the workplace, and this tendency affects physicians and workers alike.

Another issue hindering the prompt recognition of workplace morbidity, especially notable at Uretek, is limited data on chemicals. There are between 250 and 500,000 substances in use in US factories, and OSHA has exposure limits for about 500 of the total number. Part of the problem has been that the rapid proliferation of new substances has run far ahead of research on their toxic effects. In a few cases this already worrisome situation has been worsened by misrepresentation on the part of chemical manufacturers. The net result is that workers way be legally exposed to high levels of chemicals for which the health effects are not known.

Disincentives to report also impede recognition of on-the-job illness. Workers may be ignorant of the hazards, like the Uretek workers,and thus be unable to diagnose their own malady. Even a worker who feels he is sick because of his job may be ignored by his employer, at best, or risk being fired, at worst, if he or she takes the concern to OSHA. Although the OSH act provides for the anonymity of those who make reports, the instigator may be obvious in a

small shop. Less obvious is that fact that some workers may hesitate to report because they fear elimination of their job, or that they might be transferred to one that pays less. ("Dirty jobs" often provide "hazard pay".) On the employer side, there is the somewhat misplaced fear that reporting of illness in their workplace may bring OSHA inspections, fines, and higher compensation insurance premiums. All of these factors tend to keep workplace disease a silent epidemic.

One final point, though left to last, is not to be underemphasized. That is, the abysmal state of medical training in occupational medicine. Yale's curriculum includes just one hour in four years and the situation is similar, or worse at other institutions. Fewer than one third of all medical curricula include any required coursework[3] and little postgraduate exposure occurs in internal medicine or family practice residency training.[4] The consequence of this is that few physicians collect an occupational history or know what patterns to look for. As Cullen has succinctly maintained:

> Inability to recognize symptoms and signs of illness as related to work exposures precludes useful clinical intervention: opportunity for disease modification and possible prevention of disease in others is simply lost.[5]

It is to the topic of prevention that we now turn.

Preventing Workplace Disease: No More Ureteks

The threat of an OSHA inspection has been useful in motivating changes suggested by the Yale clinic in some workplaces, and OSHA itself has prompted abatement of hazards in others. As we have seen, however, this is not by itself adequate to protect worker health. The need for a separate surveillance system has been implied throughout the discussion of the Uretek case.

The debate in Connecticut, and other states, over whether the state or the federal government should run such a system is really a mirage. The real issue is what sort of agency is needed. Cullen and others have proposed that a true federal occupational health agency be created and staffed by physicians, epidemiologists, and toxicologists:

> The bottom line is that a strong OSHA needs a strong, empowered companion, a health agency that evaluates people, as OSHA evaluates machines and chemicals.[6]

Such an agency will need appropriate legal authority to compel intransigent employers to cooperate. It will need funding adequate to provide for inspection and for encouraging the training of professionals and education of workers in occupational health and risks of the workplace. It will probably want to implement exposure limits which involve biological (eg blood, urine, chest x-ray, pulmonary function tests, etc) monitoring as opposed to only airborne monitoring as done by OSHA. Biological monitoring is currently

only in use for workers exposed to lead, asbestos, coal, and cotton dust. This type of oversight would allow for better estimation of actual exposure, especially by dermal absorption, and stricter enforcement of standards. Lead workers, for example, who are found to have blood levels above the legal limit on periodic checks, cannot work until the levels fall below the safety limit, and continue to receive full pay during any furlough. The incentive is clearly very strong in those workplaces to attend to worker health. The same effect could be reinforced if workers' compensation insurance rates began to reflect the actual costs and performance of a given workplace. We have reviewed the point that only a fifth of the income losses from injuries are recouped by workers, and then only by the minority who apply for compensation.[7] Since disease represents only 1-2% of all compensation claims it makes sense to assume that very little of the actual costs are being reflected in premium rates. The bottom line is that an equitable system should account for **who benefits** and **who bears risks**, and to place the costs of disease on the former, not the latter.[8]

Perhaps most important to the success of any agency charged with protection of worker health will be reliable information concerning the incidence of disease. It is a truism that what is not yet discovered can not be altered. In order for occupational health to receive funding

commensurate with its importance, and to direct its focus once such monies are allocated, reliable statistics must be collected. The current state of affairs is so dire that there is disagreement about information as basic as the number of people who die at work in America. This is estimated to be 100,000 individuals but even this is a rough approximation. A recent study commissioned by the National Research Council excoriated the current system, especially the recent moves toward voluntary reporting, and made extensive recommendations for improvement.[9] This data will not only direct enforcement activity but also badly needed clinical research on the health effects of workplace hazards. This would include the natural history of occupational diseases; the spectrum of signs, symptoms, and laboratory findings in a specific disorder; comparisons of alternative strategies for diagnosis and treatment; development of early predictors; and assessment of the psychological and social impact of interventions requiring changes in a person's work.[10]

Larger Issues: The Ruling Paradigm

This review of the Uretek outbreak has revealed a number of themes or paradigms which undergird thinking in our society and which will stand in the way of the sorts of improvements just suggested.

The first is a "crisis orientation" not shared by other countries to the same degree. Progress in occupational health has either followed workplace disasters or been at the behest of business demands. There has been little in the way of preventive thinking. The same pattern can be seen in our parochial devotion to petroleum in a world soon to run out, to our production of nuclear and toxic waste with no safe place to put it, and in the health arena, to the sudden attention to "national" medical insurance when it has become too expensive for businesses to insure their employees.

A corollary to the crisis orientation is methodological individualism; the reduction of societal and systemic problems to the individual level. In workplace safety, the focus is on the "accident-prone" worker. With unemployment, the problem is individuals too lazy to look at the want ads. With rape it is the provocatively dressed woman. With homelessness it is disdain for living indoors. With poverty, a lack of drive and initiative. Thus it is a logical extension to view Uretek as the result of alcohol and tylenol abuse. One of the things that allows this asystemic approach to exist is the ahistorical character of American news coverage, amply exemplified in the Uretek reporting. While it is true that "newness" is newsworthiness the world over, American news coverage, with its

penchant for images and "bites", is particularly extreme in its slavishness to the "new".

Another dominant paradigm in government has been the use of economic criteria in decisionmaking. The view of the current administration has been to regulate only in instances of "market failure", and then, only after a cost-benefit analysis. There are several problems with this model when applied to workplace health. "Market failure" is practically guaranteed by "inadequate information, lack of labor mobility, unequal bargaining power, and presence of externalized costs" (eg compensation).[11] In addition, cost-benefit and effectiveness analyses raise serious ethical considerations. It is claimed that assigning costs to benefits and negative outcomes is a neutral aid to decision-making. While cost-benefit can be a useful tool, providing decision-makers with important information about the consequences of various courses of action, it becomes dangerous when it becomes <u>the</u> decision rule itself. This forces the monetization of all considerations, including ethical ones. MacLean and Sagoff, in a report commissioned for the excellent Office of Technology (OTA) report: "Preventing Illness and Injury in the Workplace", make the following point:

> How can cost-benefit analysis claim to be either neutral or comprehensive if it cannot deal with a wide range of moral, cultural, aesthetic, and political concerns? There may be some issues that raise few important cultural or moral issues; for example, the commodities markets may be left to

> determine the prices of hog bellies or potash. This does not show, however, that markets or market analysis can give us an adequate policy for public safety and health. On the contrary, where moral, political and cultural values-not simply economic ones-are at stake, we need to make moral, political, and aesthetic judgments. Cost-benefit analysis does not replace these "subjective" judgments with "objective" or "neutral" ones. Rather, it distorts or ignores the noneconomic values it cannot handle.[12]

Finally, it must be pointed out that where large differences in income, education, and access to political institutions exist, exploitation is more likely, and perhaps inevitable. This is perhaps the clearest predisposing factor...and also the most difficult to change, as it is sanctioned by beliefs at the core of American society.

These concerns might be dismissed as academic were it not for the fact that there are almost certainly many more Ureteks. Most factory owners are not so deceitful, nor do they attract attention to themselves by polluting the environment. However, this does not prevent workers from getting sick, by accident or by negligence. Most diseases have longer latency periods than the liver injury from DMF, making it difficult for worker or physician to correlate illness and exposure. As we have seen, even with these "flags" in the Uretek case, the elucidation of the outbreak was no simple matter. The paradigms discussed in this chapter are under attack and though the assault is not new they will surely be slow to perish. In the meantime there will continue to be those who critique them and offer new

ones to take their place. We have to wonder if any will do so more eloquently than Rabbi Steven Wise, speaking at a memorial service for the Triangle fire victims in 1911. In light of Uretek, and many other workplace tragedies, his words are both prophetic and incisive:

> This was not an inevitable disaster which man could neither foresee nor control. We might have foreseen it, and some of us did; we might have controlled it, but we chose not to do so. The things that are inevitable we can do no more than vainly regret, but the things that are avoidable we can effectively forestall and prevent.
>
> It is not a question of enforcement of law nor of inadequacy of law. We have the wrong kind of laws and the wrong kind of enforcement. Before insisting upon inspection and enforcement, let us lift up the industrial standards so as to make conditions worth inspecting, and, if inspected, certain to afford security to the workers. Instead if unanimity in the shirking of responsibility, we demand that departments shall cooperate in planning ahead and working for the future, with some measure of prevision and wisdom. And when we go before the legislature of the state, and demand increased appropriations in order to ensure the possibility of a sufficient number of inspectors, we will not forever be put off with the answer: We have no money.
>
> The lesson of the hour is that while property is good, life is better, that while possessions are valuable, life is priceless. The meaning of the hour is that the life of the lowliest worker in the nation is sacred and inviolable, and, if that sacred human right be violated, we shall stand adjudged and condemned before the tribunal of God and of history.[13]

Endnotes

1. Cullen (1987), B5.

2. Ibid.

3. Levy (1980).

4. Cullen (1985).

5. Ibid.

6. Cullen (1987), B5.

7. Berman (1978), 70.

8. See Ashford (1976), 358-65 for an excellent discussion of the principle of equity as it relates to workplace health. He details three principles: collective risk, voluntary choice, and randomization which are generally ignored in the literature.

9. See Pollack & Keimig, eds. (1987) for details.

10. See Cullen (1985), 513-4.

11. OTA (1985), 277.

12. Ibid, 293.

13. In Stein (1977).

BIBLIOGRAPHY

Ashford, N.A., 1976. **Crisis in the Workplace: Occupational Disease and Injury.** Cambridge, MA: MIT Press.

Bardodêj, Z. et al. 1985. Mercapturates in the Urine of Persons Exposed to N,N-Dimethylformamide. **J of Hyg Epidem Micro & Immun.** 28(2): 129-33.

Barnes, J.R. & Henry, N.W., III. 1974. The Determination of N-Methylformamide and N-Methylacetamide in Urine. **Amer Indus Hyg Assoc J.** February, 84-7.

Barnes, J.R. & Ranta, K.E. 1972. The Metabolism of DMF and DMAC. **Tox & Applied Pharm.** 23: 271-76.

Barnes, P.G. 1987. Uretek found to violate ozone standard. **New Haven Register.** 24 February, A1.

_____. 1987. State drafts tough orders for Uretek emissions. **New Haven Register.** 7 April, A1.

_____. 1987. Uretek hit with 3rd suit for waste violations. **New Haven Register.** 8 May, 7.

Beers, G.E. 1928. Compensation for Occupational Diseases. **Yale Law Journal.** 579-594.

Berman, D.M. 1978. **Death on the Job.** New York: Monthly Review Press.

Brennan, T.A. & Carter, R.F. 1985. Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals. **J of Health, Policy and Law.** 10(1): 33-80.

Butterfield, B.D. 1987. 'Chemical Sweatshop' charges worry Connecticut. **Boston Globe.** 3 March, A14.

Chary, S. 1974. DMF: A Cause of Acute Pancreatitis (letter). **Lancet.** 2(876): 356.

Chen, J.L. 1986. **Epidemiological Study on Workers Exposed to Dimethylformamide and/or Acrylonitrile.** Medical Division, E.I. du Pont de Nemours & Co., unpublished.

Cherniak, M. 1986. **The Hawk's Nest Incident: America's Worst Industrial Disaster.** New Haven: Yale University Press.

Chivers, C.P. 1978. Disulfiram Effect From Inhalation of Dimethylformamide (letter). **Lancet.** 11 Feb 1(8059): 331.

Clayton, J.W., Jr, et al. The Inhalational Toxicity of DMF. **Amer Indus Hyg Assoc J.** 24: 144-54.

Cohen, J.A. & Kaplan, M.M. 1979. The SGOT/SGPT Ratio-An Indicator of Alcoholic Liver Disease. **Dig Dis & Sciences.** November, 24(11): 835-38

Conference of State and Provincial Health Authorities. 1921-36. Report of Industrial Hygiene Committee.

Conference of State and Provincial Health Authorities. 1940. Report of Industrial Hygiene Committee.

Connecticut Acts. 1923. Chapter 93. An Act concerning reports of Occupational Disease.

_____. 1927. Chapter 326. An Act Authorizing the State Department of Health to Investigate and Make Recommendations for the Elimination or Prevention of Occupational and Making an Appropriation Therefor.

_____. 1973. 73-379. An Act Concerning an Occupational Safety and Health Act.

Connecticut General Assembly-Floor Debate. 1973. House. April 26. 4207-4257.

_____. 1973. Senate. May 9. 3012-3081.

_____. 1977. House. June 3. 5979-6001.

_____. 1977. Senate. June 8. 4204-4216.

Connecticut General Assembly-Public Hearings. 1927. Public Health and Safety Committee. February 10. 6-7.

_____. 1927. Appropriations Committee. March 16. 418-423.

_____. 1973. Labor and Industrial Relations Committee. February 9. 63-113.

_____. 1977. Labor and Industrial Relations Committee. February 22. 222-291.

Connecticut General Statutes. 1878. Sections 2505 to 2509. Authority of State Board of Health.

_____. 1920. Section 2416. Reports of Occupational Diseases.

Connecticut State Board Of Health. 1915. Untitled summary of responsiblities. Unpublished.

Coonrod, E. 1987. Dangers went unreported in sweatshop poisonings. **New Haven Independent.** 26 February, 1.

_____. 1987. Uretek neighbors: Shut it down. **New Haven Independent.** 26 March, 1.

_____. 1987. Protection is slim for Uretek Workers. **New Haven Independent.** 9 April, 9.

_____. 1987. Doctors take different sides on poisonings. **New Haven Independent.** 16 April, 12.

_____. 1987. DuPont considers cutting off factory. **New Haven Independent.** 30 April, 3.

Cullen, M.R. 1985. Occupational Medicine: A New Focus for General Internal Medicine. **Arch Intern Med.** March, 145: 511-15.

_____. 1987. Health Debacle at Uretek Not State's First, Nor Worst. **New Haven Register.** 5 July, B5.

Cullen, M.R. and Rosenstock, L. 1986. **Clinical Occupational Medicine.** Philadelphia: W.B. Saunders.

Dalich, G.M. and Larson, R.M. 1985. Temporal and Dose-Response Features of Monochlorobenzene Hepatotoxicity in Rats. **Fund & Applied Toxicology.** 5: 105-16.

De Ritis, F. et al. 1985. Serum-Transaminase Activities in Liver Disease. **Lancet.** 25 March, 685-7.

Dorsey, G. 1987. The Poison Factory. **Hartford Courant.** 14 June, Northeast Magazine, 8-28.

Dϕssing, M. 1986. Occupational Toxic Liver Damage. **Journal of Hepatology.** Vol 3, 131-5.

Ducatman, A.M. et al. 1986. Germ Cell Tumors of the Testicle among Aircraft Repairmen. **Journal of Urology.** Vol 136, October, 834-36.

Duffy, W.E. 1933. **Who's Who in Connecticut.** New York: Lewis Historical Publishing.

Franco, G. et al. 1986. Hepatotoxicity of Organic Solvents (letter). **Br J of Indus Medicine.** 43: 139.

_____. 1986. Serum Bile Acid Concentrations as a Liver Function Test in Workers Occupationally Exposed to Organic Solvents. **Int Arch Occup Environ Health.** 58:157-64.

Gray, A.S. 1935 Importance of Industrial Hygiene. **JAMA.** V.105, October 12, 1157-1160.

_____. 1928. Occupational Disease Prevention in Connecticut. **Connecticut Industry.** September.

Grillo, A.E. 1964. Fifty Years of Workmen's Compensation-An Historical Review. **Connecticut Bar Journal.** V. 38, 239-251.

Hamm, S. 1987. Hepatitis 'outbreak' at Uretek. **New Haven Journal Courier.** 19 February, A1.

_____. 1987. OSHA promises to 'get to bottom' of Uretek woes. **New Haven Register.** 25 February, A1.

_____. 1987. City, OSHA disagree on Uretek illness tip. **New Haven Register.** 27 February, A1.

_____. 1987. Uretek strike settled, safety key issue. **New Haven Register.** 30 May, A1.

Hartford Times. 1922. Dr Black leaves Health Department, Osborn Successor. 6 November.

Hartford Courant. The Mess at Uretek. 6 March, B10.

Hirschfield, N. 1987. The Poison Plant. **NY Daily News Magazine.** 15 March, 22-5.

Kauffman, M. 1987. Uretek accused of stalling. **Hartford Courant.** 8 March.

Kennedy, G.L., Jr. et al. Acute and Subchronic Toxicity of Dimethylformamide and Dimethylacetamide Following Various Routes of Administration. **Drug and Chem. Toxicology.** 9(2): 147-170.

Kitka, W. 1987. Sick Days at Uretek. **Connecticut.** August, 62-7.

Krivanek, N.D. et al. 1978. Monomethylformamide Levels in Human Urine After Repetitive Exposure to DMF Vapor. **J of Occupational Medicine.** March 20(3): 179-82.

_____. 1978. MMF Levels in Human Urine After Repetitive Exposure to DMF Vapor. **J of Occup Med.** March, 20(3): 179-82.

Levin, S.M. et al. 1987. Testicular Cancer in Leather Tanners Exposed to Dimethyl Formamide (DMF)(letter). **Lancet.** November 14.

Levy, B. and Wegman, D. eds., 1983. **Occupational Health.** Boston: Little Brown.

Lundberg, I. et al. 1981. Some Observations on Dimethylformamide Toxicity. **Toxicology.** 22: 1-7.

_____. 1985. Normal Serum Activities of Liver Enzymes in Swedish Paint Industry workers with Heavy Exposure to Organic Solvents. **Brit J Industr Med.** 42: 596-600.

_____. 1986. Relative Hepatotoxicity of Some Industrial Solvents after Intraperitoneal Injection or Inhalation Exposure in Rats. **Environ Research.** 40: 411-20.

Lyle, W.H. 1979. DMF and Alcohol Intolerance. **Br J of Indus Med.** 36: 63-6.

Mandate for Leadership. 1981. The Occupational Safety and Health Administration. Washington: Heritage Foundation, 454-60.

Massman, W. 1956. Toxicological Investigations on Dimethylformamide. **Brit J Industr Med.** 13: 51-4.

Maxfield, M.E. et al. 1975. Urinary Excretion of Metabolite Following Experimental Human Exposures to DMF or to DMAC. **J of Occup Med.** August, 17(8): 506-11.

McGraw, M. 1987. Federal doctor to investigate Uretek Illnesses. **Hartford Courant.** 4 March, D5.

_____. 1987. OSHA slipping as work-place watchdog. **Hartford Courant.** 8 March, B1.

_____. 1987. OSHA sending reinforcements to state. **Hartford Courant.** 12 March, A16.

McGraw, M. & Kauffman, M. 1987. Records say OSHA knew of possible Uretek danger. **Hartford Courant.** 10 April,A1.

Mitchell, J.W. 1987. Screening for Hepatic Damage. **J of Occup Med.** March, 29(3), 266-7.

NASW News. 1987. Social Worker Champions Plant Workers. October.

Navarro, V and Berman, D., eds. 1983. **Health and Work Under Capitalism.** New York: Baywood Publishing Company.

Occupational Health & Safety. 1987. Uretek cited for Violations. 18.

Occupational Safety and Health Act of 1970. Public Law 91-596. 29 December.

Office of Technology Assessment. 1985. **Preventing Illness and Injury in the Workplace.** Washington, DC: U.S. Congress, OTA-H-256.

Osborn, S.H. & Johnston, J.H. 1952. Occupational Health Program in a State Health Department. Presented at APHA, October 23.

Pollack, E.S. and Keimig, D.G., eds. 1987. **Counting Injuries and Illnesses in the Workplace: Proposals for a Better System.** Washington, DC: National Academy Press.

Potter, H.P. 1973. Dimethylformamide-Induced Abdominal Pain and Liver Injury. **Arch Environ Health.** Nov 27: 340-1.

_____. 1974. Toxicity of DMF (letter). **Lancet.** 2 November, 2(7888): 1084.

Povinelli, N. 1987. Uretek found guilty for illegal storing of hazardous wastes. **New Haven Register.** 3 February, A1.

_____. 1987. Uretek flap gives trial judge pause. **New Haven Journal-Courier.** 26 February, A1.

_____. 1987. Uretek is fined $255,000 for waste violation. **New Haven Register.** 14 April, A1.

Ramazzini, B. 1964. **Diseases of Workers (De Morbis Artificum).** New York: Hafner Publishing Co.

Ravo, N. 1987. At factory, Chemical fumes set off strike. **New York Times.** 13 April, B1.

Redlich, C.A. et al. In Press. Liver Disease Associated with Occupational Exposure to the Solvent DMF. **Arch Int Med.**

_____. 1987. Outbreak of Occupational Hepatitis - Connecticut. **MMWR.** 27 February, 36(7) reprinted in **JAMA.** 20 March, 257(11): 1453.

Report of the president to Congress on Occupational Safety and Health for Calender 1985. June 1986. Washington: US Government Printing Office.

Santodonato, J. et al. 1985 **Monograph on Human Exposure to Chemicals in Workplace: Dimethylformamide.** Prepared for National Cancer Institute by Syracuse Research Corporation, NY.

Sidel, V. and Sidel, R. 1984. **Reforming Medicine: lessons of the last quarter century.** New York: Pantheon Books.

Smith, R.S. 1976. **The Occupational Safety and Health Act: Its Goals and Achievements.** Washington, DC: American Enterprise Institute.

Stein, Leon, ed. 1977. **Out of the Sweatshop: The Struggle for Industrial Democracy.** New York: Quadrangle/The New York Times Book Co..

Tolot, F. et al. 1957. Intoxication par Diméthylformamide. **Proceedings: Société de Médecine et D'Hygiène du Travail.** 21 December, 602-6.

_____. 1968. Intoxication par Dimethylformamide. **Proceedings: Société du Médecine du Travail et D'Ergonomie de Lyon.** 12 January 714-17.

Ungar, H. et al. 1976. Acute and Protracted Changes in the Liver of Syrian Hamsters Induced by a Single Dose of Aflatoxin B_1. Observations on Pathological Effects of the Solvent DMF. **Br J Exp Path.** 57: 157-64.

US Department of Labor. 1986. **Occupational Injuries and Illnesses in the USA by Industry, 1984.** Bulletin #2259. Washington: Bureau of Labor Statistics.

_____. 1986. **BLS Reports on Survey of Occupational Injuries and Illnesses in 1985.** Washington: Bureau of Labor Statistics.

_____. 1987. **BLS Reports on Survey of Occupational Injuries and Illnesses in 1986.** Washington: Bureau of Labor Statistics.

US Department of Labor/OSHA. 1979. **Lost in the Workplace: Is there an occupational disease epidemic?.** Washington: US Government Printing Office.

Verhoeff, A.P. 1987. Organic Solvents in the Indoor Air of Two Small Factories and Surrounding Houses. **Int Arch Occup Environ Health.** 59: 153-63.

Waldron, H.A. 1982. Solvent Exposure and Liver Function (letter). **Lancet.** 4 Dec 2(8310): 1276.

Weindling, P. 1985. **The Social History of Occupational Medicine.** London: Croom Helm.

Wiles, J.S. and Narcisse, J.K., Jr. 1971. The Acute Toxicity of Dimethylformamides in Several Animal Species. **Amer Indus Hygiene Assoc J.** August, 539-45.

Zimmerman, H.J. 1978. **Hepatotoxicity.** New York: Appleton-Century-Crofts.

YALE MEDICAL LIBRARY

Manuscript Theses

Unpublished theses submitted for the Master's and Doctor's degrees and deposited in the Yale Medical Library are to be used only with due regard to the rights of the authors. Bibliographical references may be noted, but passages must not be copied without permission of the authors, and without proper credit being given in subsequent written or published work.

This thesis by has been used by the following persons, whose signatures attest their acceptance of the above restrictions.

NAME AND ADDRESS	DATE